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General Surgery

Collective Review of Hydatid Disease

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IN most cattle-breeding countries hydatid disease is common. In some of these countries it is much more common than the responsible Public Health authorities realize, who do not even realize the seriousness of the disease itself. The prevention of hydatid disease is simpler than that of many other diseases, yet in many countries not much is done to eradicate it, though it could be done by destroying stray dogs; teaching the owners of pet dogs not to feed them anything raw in the form of meat, and instructing the butchers not to feed with "bad meat," which is often a hydatid cyst full of hundreds and thousands of scolices, the dogs that regularly attend the slaughtering ceremonies in many a butcher's shop in some of these countries.

That hydatid disease can be a serious condition in many cases, causing a great deal of incapacity, suffering and not too infrequently death, is well known. With the enlargement of the scope of surgery, the seriousness of hydatid disease has become evident. It is now known that not only can the liver, the lungs, the spleen or the kidney be a site of hydatid disease, but the orbit, the thyroid gland, the brain and the heart can also harbor hydatid cysts, with devastating and often fatal results. A patient can sometimes harbor scores and even hundreds of hydatid cysts in the liver and lungs. That such a patient is doomed to die is only too evident, and I have encountered such patients.

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The Parasite. — Taenia echinoccocus is the smallest of the cestodes. The mature worm measures 5 mm. in length and 0.5 mm. in width at its widest part. It occurs in great numbers in the intestines of the infested dog, which is its final host. It is similarly found in the intestines of jackals, wolves and foxes. The worm is made up of 4 segments ("proglotids"). The first segment contains the head and the neck. The head has at its vertex a retractile prostellum with a double row of about 40 hooklets, and behind the hooklets there are 4 suckers by means of which it attaches itself to the intestinal mucosa of the dog and gets its nutrition. It takes eight weeks to mature in the intestines of the dog. Its life span is five months, after which it dies. During its life span the last (fourth) segment becomes separated from the worm. The segment usually contains scores of ova. These are excreted with the stools. It is apparent, therefore, that an infested dog can excrete many thousands of ova daily. These ova have a chitinous outside cover. They are highly resistant to heat, cold, dryness or moisture. These ova pass on to the hands of people who own dogs, or they are spread over grass and vegetables, or they may contaminate the water supply. It is when human beings or sheep or cattle swallow these ova that infestation takes place.

Once the ovum is swallowed by man, sheep or cow, the outer chitinous membrane is dissolved by the gastric or duodenal juices and a small microscopic hexacant embryo is hatched, which by its six sharp spiculae bores its way through the mucosa and gets into the radicles of the portal circulation. Once it has traversed the mucosa it loses its 6 spicules and is carried as an embolus in the blood stream of the portal circulation to the liver. Obviously a person can swallow one or many ova at the same time. One of my patients, a child 6 years old, was seen by his mother, when he was 2 years old, eating the feces of a dog. This child, on whom I operated when he was 6 years of age, had hundreds of cysts in his liver and many in his lungs. Once the embryo reaches the liver it is usually caught there. As the right lobe of the liver has a larger supply than the left lobe, it receives more of these embryos. Clinically one observes many more hydatid cysts of the right lobe than of the left. The liver is thus the first sieve for these embryos.

Some embryos, however, do escape from this first obstacle and go to the lungs, where they may settle. Hence the lungs are the second sieve. If they escape the lungs too, then they may go to the left side of the heart and from there to the general circulation. Once they get into the general circulation, they may settle in any organ or tissue of the body.

When the embryo reaches its final destination—the liver, for example—it begins to take the cystic form. Very early the tissues of the organ react in an attempt to limit extension of the cyst. This is the beginning of the larval stage. The barrier put out by the organ is of fibrous tissue and is called the ectocyst. The ectocyst is part of the host, not part of the parasite. In the course of time, once the cyst has been completely removed, the ectocyst disappears completely. The parasite itself, in its second stage in the organ of its intermediate host (man, sheep or cattle), grows slowly. It is made up of two layers, an externally laminated layer and a much thinner inner germinal or granular layer; hence its name, Echinoccocus granulosus. It is this germinal layer that produces the brood capsules and daughter cysts.

The cyst grows so slowly that in some cases of hydatid cyst of the liver the cyst may be almost as old as the patient, infestation having taken place in infancy. It is said that a cyst grows to be 2 mm. in

diameter by the end of the third month. In some sites, however, such as the bones or the brain, it becomes clinically evident much earlier. Some cysts attain tremendous size; the largest reported in the literature contained 11 liters of fluid. The largest cyst in my series was an infected cyst of the liver containing 7 liters of infected hydatid fluid. Hundreds of daughter cysts were present.

The hydatid fluid is crystal clear. If it is allowed to stand still the scolices, numbering many thousands in a mature cyst, settle down to the bottom. This is the so-called hydatid sand. The scolex has a dual potentiality. If it escapes from the cyst, e. g., during an operation, and remains in the host, it is capable of forming a secondary cyst in the same intermediate host; hence one of the dangers of allowing hydatid fluid to escape the tissues of the host during an operation.

When a scolex gains entrance into the gastrointestinal tract of a dog, it develops into a mature T. echinoccocus in the dog's intestine. When a dog eats one mature fertile cyst he ingests hundreds of thousands of scolices; hence hundreds of thousands of T. echinoccocus develop in the intestines. If the hydatid fluid escapes into the peritoneal or pleural cavities during an operation, it can give rise to serious anaphylactic shock. In one of my cases, fluid from a hydatid cyst of the liver accidentally escaped into the general peritoneal cavity during the operation, and instantaneous cardiac arrest occurred. I had to open the chest in the left fourth intercostal space and massage the heart, which within three and one-half minutes began to function. The patient recovered and left the hospital in good condition.

General Symptoms.—In countries where hydatid disease is common, as in Lebanon, the possibility of a hydatid cyst is considered in the differential diagnosis of any tumefaction. Even in patients with increased intracranial tension and the general symptoms of brain tumor the possibility of hydatid cyst of the brain must be considered. A patient with a cystic mass in the abdomen, especially one connected with the liver, makes the surgeon first think of hydatid cyst of the liver. If a paitent says that he has had recurrent attacks of urticaria, the possibility of a hydatid cyst somewhere in the body immediately presents itself.

Laboratory examinations such as Cassoni' Cuti reaction, Weinberg's complement fixation test and eosinophilia when positive, help to confirm our diagnosis, but negative results do not mean much. In several cases the presence of hydatid cysts has been confirmed by operation after all of these tests had given negative results. Roentgen examination is helpful; often the results are characteristic and make the diagnosis certain. A cystic round mass in connection with the liver, with or without some calcification, is a fairly sure sign of hydatid cyst of the liver. A smooth, regular, rounded localized elevation of the diaphragm toward the thorax is characteristic. A regular, clear round shadow in the lung is almost pathognomonic. Occasionally a crescent-shaped shadow in the upper portion of the cystic mass in the lung, due to the presence of air outside the endocyst but inside the ectocyst, is characteristic. An infected cyst of the lung when the endocyst is separated from the ectocyst with the ectocyst occupying only the lower part of the cyst cavity, results in an irregular smooth line in the lower half of the cyst cavity on the roentgenogram. The so-called water-lily appearance is typical of a ruptured hydatid cyst.

Hydatid Cyst of the Liver.—The liver is the commonest site of hydatid cyst formation. Statistics show it to be involved in 60 to 80 per cent of the cases. In my series 52 per cent of the hydatid cysts were in the liver. In this organ the hydatid cyst

grows slowly but may reach an extremely large size. It may be quiescent; the patient may not even be aware of a relatively large hepatic cyst until the physician detects it during a routine physical or roentgen examination. The latter may reveal a regular, rounded, localized elevation of the diaphragm. In other cases the patient himself may feel a mass in the right upper quadrant of the abdomen and come to consult a doctor. In still other cases there may be definite complaints, such as distention, a sense of fullness, indigestion or urticaria; or the cyst may rupture into the biliary passages and cause severe pain resembling biliary colic. The gallbladder may be greatly distended after such a perforation, and clinically the condition may suggest empyema of the gallbladder. If a hepatic cyst becomes infected, the picture may be that of abscess of the liver.

The infected cyst may perforate subdiaphragmatically, presenting the clinical picture of subdiaphragmatic abscess. A cyst in the dome of the liver may perforate into the chest and cause collection in the pleural cavity or it may even perforate into the lung and the patient may cough out the daughter cysts and even the endocyst that has come from the dome of the liver. If the cyst perforates into the pleural cavity or into the peritoneal cavity, it may kill the patient by anaphylactic shock. If the patient overcomes this shock, the thousands of scolices in the escaped fluid may develop into as many hydatid cysts in the course of time and, like metastases from a malignant tumor, kill the patient in due course. Sometimes a cyst of the liver may gradually become calcified. A calcified cyst is usually a dead cyst.

Treatment: Never should any exploratory puncture of a cystic mass be done, especially in countries where hydatid disease is common. The fluid in a hydatid cyst is always under great tension, and, no matter how small a needle is used, the fluid will leak out from the puncture hole. This escape of fluid may kill the patient from anaphylactic shock, and, even if the patient survives the shock, hundreds of scolices will have been thrown into the peritoneal cavity, each one of which in time will become a hydatid cyst. In an operation for hydatid cyst of the liver, after the peritoneal cavity has been opened, the cyst should be entirely surrounded with pads. The cyst is then punctured with a thick two-valved needle, all its fluid contents being aspirated directly into a bottle. Then, through the second valve, absolute alcohol is aspirated and injected into the cyst cavity. After four or five minutes the injected alcohol is aspirated. (Forty per cent solution of formaldehyde could be used instead of alcohol.) The purpose of this injection of alcohol or formaldehyde is to damage as much as possible the scolices that are left in the cavity of the ectocyst, so that if any remain or escape they will not develop into secondary hydatid cysts.

After all fluid has been aspirated from the cyst, the ectocyst is incised for a length of 3 or 4 cm. and the endocyst is removed completely, with all its daughter cysts. No part of the endocyst should be left in. The cavity of the ectocyst is thoroughly washed with physiologic solution of sodium chloride and aspirated. The walls of the ecocyst are next plicated as much as possible to diminish the empty space, and the incision into the ectocyst is closed tightly.

Before the advent of antibiotics I used to drain the large cavity, which meant several months of drainage. Since the advent of antibiotics, however, I have been closing all cysts without drainage with good results. My cases of hydatid cyst of the liver since 1935 number 178.

A cyst in the dome of the liver is best approached through the chest. It is a direct approach and makes the operation

considerably easier. I have operated in 12 such cases, with very satisfactory results.

If a cyst has ruptured into the biliary passages and the gallbladder is distended, a cholecystectomy should be done. The mother cyst should be located in the liver, and the endocyst and all daughter cysts should be removed. The common bile duct should be opened and explored; if any daughter cysts are present they should be removed. The duct should be irrigated and T tube drainage installed for ten to fifteen days. I have operated for this condition in 4 cases at the American University Hospital.

If a hydatid cyst of the liver ruptures into the peritoneal cavity before the patient is operated upon and if the patient survives the anaphylactic shock, he should be operated upon as soon as he recovers from the shock and can stand an exploratory laparotomy. The endocyst should be removed completely, with all its daughter cysts, and the peritoneal cavity should be washed and thoroughly irrigated with several liters of saline solution to remove as many of the scolices as possible. The following case demonstrates the seriousness of the condition when a cyst ruptures into the peritoneal cavity:

A woman, 28 years old, had a hydatid cyst of the right lung (upper lobe) and a hydatid cyst of the liver. She was operated upon for the pulmonary cyst, made a satisfactory recovery and left the hospital. Eight months later she was readmitted for treatment of the cyst in the liver. During the operation, by accident, the cyst ruptured, and before precautions could be taken the hydatid fluid escaped into the peritoneal cavity. Almost instantaneously she had cardiac arrest. Without loss of time, the left fourth intercostal space was opened and massage of the heart started. The heartbeat came back quickly, within three and one-half minutes after the arrest. The chest was closed and the operation for removal of the hydatid cyst and irrigation of the peritoneal cavity completed.

A calcified cyst of the liver should not be operated upon unless it is giving rise to pressure symptoms. Always, before closing up the abdominal cavity, one should explore the other parts of the liver, the spleen and the rest of the abdomen to to make sure that there are no other cysts.

I have had 178 cases of hydatid cyst of the liver; the largest cyst contained 7 liters of infected fluid. In 70 cases more than 1 cyst was present; in 105 more than 5 cysts, and in 1 there were hundreds of cysts in the liver.

The Lungs.—The second commonest site for hydatid cyst is the lung. I have had 104 cases of pulmonary hydatid cyst. Here again there may be only one cyst or there may be several. One or both lungs may be involved. The patient may present no symptoms at all, and the cyst or cysts may be detected only on routine roentgen examination of the chest, but in other cases the patient may have a cough or expectoration, the sputum being sometimes tinged with blood. The cyst may open to a bronchus and the patient may cough out the hydatid fluid, the daughter cysts and sometimes even the endocyst, thus curing himself. Such cures, however, are not frequent. If the cyst is large and it ruptures, the patient may drown in the hydatid fluid. It may rupture into the pleural cavity and cause severe and sometimes fatal anaphylactic shock. The cyst may become infected, and the patient then presents a typical picture of pulmonary abscess. Roentgen study is helpful as it shows the size, shape, number and location of the cysts. The shadow of each cyst is round, with a smooth border, and in many cases almost a perfect circle. When air has escaped into the ectocyst cavity, outside the endocyst, the reversed crescent shadow in the upper part of the cyst is characteristic. If an infected cyst opens to the outside through a bronchiole, the roentgen rays will show the cyst and the collapsed endocyst by a wavy horizontal line somewhere near the middle of the

cyst (the so-called water-lily appearance).

Treatment is by operation. The chest is opened under intratracheal pressure anesthesia; the cyst is identified, and pads are placed all around it. With the lung in the collapsed state an incision 2 to 6 cm. long is made carefully through the ectocyst, its length depending on the size of the cyst. Then the anesthetist is asked to expand the lung. The endocyst usually spurts out and is collected into a small receptacle held in readiness. After removal of the cyst, one should always look inside the ectocyst cavity and locate the openings of the small bronchioles that are almost invariably found therein. These should be identified and closed. The ectocyst is then obliterated as far as possible. The pleural cavity is drained for two or three days by means of a Foley catheter under water. If there are multiple cysts in the lung they can be removed all in one stage, if there are not too many.

One of my patients, a youth 19 years old, had 7 cysts in the left lung and 5 in the right. He was operated upon twice. During the first operation the 7 cysts were removed from the left lung, and six months later, at another operation, the 5 cysts were removed from the right lung. In both instances he made an uneventful recovery.

Unless a cyst is badly infected and has destroyed a lobe of the lung lobectomy is not necessary.

Seventy of my patients had single cysts; 33 had more than 1 cyst, and 1 had 7 in the left and 5 in the right lung.

The Spleen.—Hydatid cyst of the spleen is less frequent. I have encountered 15 cases. In the great majority these cysts are asymptomatic. A cystic mass may be palpable in the left upper abdominal quadrant on physical examination, or a routine roentgen examination may show a round shadow in the splenic area or an elevation of the diaphragm. Hydatid cyst of the spleen may be infected. The clinical signs will be then those of splenic abscess.

The treatment of a large hydatid cyst of the spleen is splenectomy, which can be done by the abdominal route in most cases. In cases of difficult and large cysts, however, an excellent approach is the thoracoabdominal route.

The Pancreas.—Hydatid pancreatic cyst is rare. I have observed no cases. A pancreatic cyst is difficult to diagnose before operation as a hydatid cyst. In countries where hydatid cysts are common, however, one can suspect it, particularly if the patient has had recurrent attacks of urticaria and has a positive Cassoni reaction, and a "positive Weinberg" and eosinophilia. Clinically, hydatid cyst of the pancreas will show a tumefaction and may cause pressure on the biliary passages, if it is large and located in the pancreatic head.

Treatment consists in removing the endocyst with the usual precautions. Before closing the abdomen it is necessary to explore the other abdominal organs for additional hydatid cysts.

The Uterus.—In the uterus these cysts are exceedingly rare. Augusto Turenne of Uruguay reported 10 cases in 1918. The clinical picture is not characteristic at all, and it is almost impossible to diagnose before operation.

Treatment consists in removal of the cyst with the usual precautions, always remembering that other organs in the body may also harbor hydatid cysts.

The Kidney.—The kidney may harbor hydatid cysts, and this is not a rarity. I have had 18 cases of hydatid cyst of the kidneys. A hydatid cyst of the kidney may cause no symptoms at all; it may be found only by a routine examination, as when a mass is palpated in the renal region, or a routine roentgenogram may show the cystic mass in connection with

the kidney. In some cases, however, there may be pain in the region of the kidney, or renal function may be diminished. If the cyst becomes infected, the symptoms are those of renal abscess. In some cases the cyst may rupture into the pelvis or into one of the calices of the kidney, giving rise to severe renal colic. In such case the patient may pass daughter cysts with the urine; even pieces of the endocyst may come out on micturition.

Treatment of hydatid cyst of the kidney depends on how much of the involved kidney has been destroyed and whether the opposite kidney has good function. If the cyst has involved only a portion of the kidney and is protruding outward from the kidney, the cyst only can be removed or a partial nephrectomy done. If it has damaged most of the kidney a nephrectomy is in order, after one has made sure that the function of the opposite kidney is good. In such a case one must always make sure that the patient does not harbor other cysts, especially in the liver and the lungs.

The Heart.—Hydatid cyst of the heart is rare, and in almost all the cases it is diagnosed only at autopsy. A hydatid cyst of the heart may be primary or secondary. A primary cyst results when the hexacant embryo has escaped the hepatic and pulmonary sieves. There are no special pathognomonic symptoms and many hydatid cysts of the heart are silent yet have caused sudden death on rupture. In some cases murmurs have been present, but these have not been pathognomonic.

In 1846 Criensinger collected 15 cases of hydatid cyst of the heart. In 1868 Oisterin collected 21 cases, and in 1905 O. G. Grulee reported 55 cases, including 1 of his own. From the cases reported it is evident that hydatid cyst of the heart is usually embedded wholly or partly in the cardiac wall. The size of the cyst varies, in the autopsy reports, from that of a cherry to that of an orange, but the majority have been 3 to 4 cm. in diameter. In 70 per cent of the reported cases there was only 1 cyst in the heart. Usually the heart, like other organs, forms a connective tissue envelope around the cyst. A hydatid cyst of the heart, because of the continuous heartbeats, usually ruptures early. This rupture may be one of two kinds, primary or secondary. In a primary rupture, when the cyst is still very small, the patient may survive the associated anaphylaxis. From this rupture secondary cysts occur in the lungs or the brain, depending on whether the rupture is in the right or left heart. As time passes after the first rupture, the mother cyst may again grow larger and daughter cysts develop. When such a cyst ruptures it causes sudden death from massive embolisms formed by the endocyst and the daughter cysts.

In 1916, according to Deve, the number of reported cases of hydatid cyst of the heart had risen to 105. At the American University hospital there was 1 patient with hydatid cyst of the heart who died; the cyst was first discovered at autopsy. On gross pathologic examination it was reported that the cyst was lined by a chitinous membrane and contained clear fluid, in which scolices and hooklets were found. The anterior descending branch of the left coronary artery coursed beneath the cyst. Its upper part was calcified and greatly narrowed below the cyst. The myocardium of the left ventricle showed areas of scarring, to which thrombi were attached. Microscopic examination revealed that the cyst rested between the visceral pericardium and the myocardium. The anterior descending branch on the left coronary artery was calcified. There was a granulating hemorrhagic area adjacent to an atheromatous patch, and the lower part of the lumen contained fresh thrombi. In the left ventricle, the anterolateral wall

showed areas of acute necrosis of the myocardium, with mild leukocytic infiltration, together with areas of fibrous scar tissue on the endocardial aspect, to which thrombi were attached. The right ventricle showed small foci of fibrosis in the myocardium. Microscopic study of the conduction system revealed no special lesions. Examination of the abdominal contents revealed passive congestion of the liver, spleen and kidneys. No other hydatid cysts were detected in the body (the skull was not opened).

In all countries where hydatid disease is common, the possibility of a hydatid cyst of the heart should be kept in mind, especially with patients who have multiple hydatid cysts of the lungs. Special roentgen studies of the heart should then be done. With the present rapid advances in thoracic and cardiac surgery, sudden death in many cases could be prevented if the condition were diagnosed, the patient operated upon and the hydatid cyst removed.

The Brain.—There have been 4 cases of hydatid cyst of the brain at the American University Hospital in Beirut. The lesion is not a rarity, and it may be primary or secondary. When it is primary, the hexacant embryo must have passed the liver and lung barriers and proceeded to the brain by the arterial system. Secondary cysts of the brain occur from a rupture of a hydatid cyst of the left side of the heart containing scolices. Of course, many a patient may die after such a rupture. The frontal lobe is the most frequent site of the cyst.

Owing to the softness of the cerebral tissues the cyst grows rather rapidly, and protective reaction, "ectocyst formation," is usually minimal. As the cyst grows, causing pressure, the patient begins to show signs and symptoms of increased intracranial tension. A primary cyst of the brain is most frequent in children (80 per cent). In a child, such a cyst may grow to quite a large size. Harold Dew reported a hydatid cyst of the brain that contained 20 ounces of fluid. This, of course, was due to the relative softness of the skull. A primary cyst of the brain is usually single, and, since it is usually not mature at the time of operation, it contains no daughter cysts.

Treatment is by operative removal. The usual precautions for preventing the escape of fluid, of course, should be taken. The results of the operation depend on the location of the cysts. It is an easy procedure when it is superficial. Secondary cysts of the brain are always secondary to intracardiac rupture into the left side of the heart, from which the scolices may be carried through the aorta to any part of the body and the brain as well. Multiple cysts of the brain, all about the same size, may therefore be present, and they may be on either the right or left half of the brain, or both. In such cases one should always look for cysts in other parts of the body. Of course a patient with hydatid cyst of the brain may die of increased intracranial tension if early operation is not performed.

Thyroid Gland. — Hydatid cyst of the thyroid gland is not common, but at the American University Hospital there have been 3 cases. In all 3 there was localized enlargement of the thyroid gland. The basal metabolic rates were normal. The preoperative diagnosis was adenoma of the thyroid gland, but at operation hydatid cysts were revealed. It is therefore wise to remember, in cases of cystic swellings of the thyroid gland in countries where hydatid disease is common, the possibility of hydatid cyst of this gland.

Treatment is removal of the cyst and thorough examination of the patient's other organs to see whether there are cysts in other parts of the body.

SUMMARY

Hydatid disease, which is highly prevalent in cattle-breeding countries, is here made the subject of a comprehensive review which covers briefly the life cycle of the parasite and proceeds to a discussion of the symptoms, sites and treatment. Illustrative cases are described.

RESUMEN

Le enfermedad hidatídica, de gran importancia en los países ganaderos, es estudiada en este artícula en una revisión que se extiende brevemente sobre el ciclo vital del parásito, y discute los síntomas, localizaciones y tratamiento. Se describen varios casos demostrativos.

RIASSUNTO

L'idatidosi, la cui frequenza aumenta nei Paesi ricchi di bestiame, viene fatta oggetto in questo lavoro di uno studio che riguarda il ciclo di vita del parassita, la sintomatologia, le sede e la terapia. Vengono descritti casi dimostrativi.

RÉSUMÉ

L'auteur présente une étude du kyste hydatique, particulièrement fréquent dans les régions d'élevage. Il rappelle brièvement le cycle de vie du parasite, puis discute les symptômes, les localisations et le traitement. Quelques exemples sont décrits.

ZUSAMMENFASSUNG

Die Echinokokkenkrankheit, die in Ländern, wo Rinder gezüchtet werden, häufig vorkommt, wird hier einem umfangreichen Studium unterworfen, das sich mit dem Entwicklungszyklus des Parasiten, mit den Krankheitserscheinungen, dem Sitz der Erkrankung und der Behandlung beschäftigt. Erläuternde Krankheitsfälle werden beschrieben.

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Intestinal Obstruction Caused by Spontaneous Rupture of the Ileum, with Volvulus Due to Abscess Formation

Report of An Unusual Case

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An obese white woman aged 75 was admitted to my service at the Jamaica Hospital on June 16, 1955. Her chief complaints were severe abdominal pain and cramps in the lower part of the abdomen for the past four days, with vomiting. On the day of admission the patient vomited several times. She stated that for the past four days she had been taking cathartics and enemas, despite which she had had no bowel movements.

History. — In 1950 the patient underwent a vaginal hysterectomy performed for complete procidentia. In 1951 an esophageal diverticulum was resected.

Physical Examination. — The patient was dehydrated and in acute pain. The skin was dry. The pulse rate was rapid and the blood pressure in millimeters of mercury was 180 systolic and 92 diastolic. Marked tenderness and spasm were noted in the right lower abdominal quadrant, and a large mass was palpable in this region. Dullness on percussion was elicited over the mass, extending from the midline and below the umbilicus and to the crest of the ilium. Spasm also was noted in the right hypochondrium. The erythrocyte count was 4,630,000 per cubic millimeter of blood, with 90 per cent hemoglobin; the leukocyte count 7700 with 62 per cent polymorphonuclear cells and 28 per cent lymphocytes. No rods were present. The specific gravity of the urine was 1.020. There was a 4 plus reaction for sugar, a trace of albumin and no acetone. The value for blood sugar was 258 mg. per hundred cubic centimeters. A roentgen flat plate taken on admission revealed no obstruction.

On June 17 a high colonic enema was given, and a copious bowel movement resulted. On June 18, abdominal cramps persisted in spite of the bowel movement. Vaginal examination was done, revealing a large globular mass palpable in the pelvis, on the right side. On June 19, the patient had spontaneous bowel movement and expelled copious amounts of gas; no cramps were reported. The diabetes was under control. On June 20 the mass in the abdomen disappeared. The abdomen was soft. The patient had been having bowel movement. A flat roentgen plate and barium enema study revealed no signs of obstruction or distention. No masses were visualized. The patient was discharged, improved, with a diagnosis of partial intestinal obstruction due to fecal impaction.

On December 17, six months after her first admission to the hospital, the patient was readmitted with severe abdominal pain, which had started as slight cramps

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two weeks earlier. She had noticed a lump the size of an orange in the right upper quadrant of the abdomen. Two days prior to admission the cramps grew more severe and the mass increased in size. There was no vomiting. The patient stated that her bowel movements had been extremely irregular and that she was constipated most of the time. On this admission the erythrocyte count was 2,560,000 per cubic millimeter of blood, with 50 per cent hemoglobin; the leukocyte count was 17,100, with 82 per cent polymorphonuclears, 77 per cent segmental cells, 12 per cent lymphocytes and 5 rods. The value for blood sugar was 124 mg. per hundred cubic centimeters. On December 18, the day after readmission, the size of the mass increased three times in twenty-four hours. The patient appeared toxic and acutely ill. In view of the rapid increase in size of the mass in the right upper abdominal quadrant, I concluded that this was a carcinoma of the transverse portion of the colon which had ruptured and formed an abscess. Immediate exploration was advised and performed.

Operative Picture.—A longitudinal incision was made over the mass in the right upper quadrant of the abdomen. A small incision was made into the peritoneal cavity, and a large amount of foul-smelling, thick, purulent discharge escaped (approximately 750 cc.). The incision in the peritoneum was lengthened, and a large abscess cavity was observed, containing a mass of bowel with a large perforation in it. The perforation would admit a thumb with ease. It was difficult to determine by inspection whether this was large or small bowel. By sharp and blunt dissection, the transverse portion of the colon was revealed to be intact and adherent to this mass. The colon was separated and freed. The perforation was in a large mass sitting on top of a loop of ileum, which was gangrenous owing to a volvulus. The gangrenous loop of bowel with the mass on its upper third was resected and an end-to-end anastomosis performed. It appeared to be an adherent mass or part of a loop of ileum. The peritoneal cavity was drained by means of a cigarette drain placed in the abscess cavity and the abdomen was closed in layers with interrupted No. 1 chromic sutures. The patient had a stormy course for five days, with profuse purulent drainage from the wound, which gradually subsided. The patient made a smooth recovery thereafter and was discharged from the hospital on January 3, two weeks after readmission.

Pathologic Report.—In the middle third of the resected bowel there was a firm, irregular pedunculated mass measuring 11

Schematic drawing of volvulus.

by 7 by 5 cm. The pedicle was small and twisted. A large necrotic cystic area of erosion through this pedunculated mass was suggestive of tumor tissue or fat necrosis. Numerous large giant cells of foreign body reaction type were observed microscopically.

CONCLUSIONS

The author reports a case which, in his opinion, is an instance of spontaneous rupture of the small bowel with abscess formation into a fat pad on the ileum. It had occurred, apparently, six months earlier, at the first attack. As the abscess in the fat pad grew larger during a six-month period, the omentum wrapped itself around it, thus localizing the lesion. Also, as the abscess grew larger it twisted on its pedicle, producing torsion and volvulus of a loop of ileum.

This case is reported to call attention to a rare mechanism by which a loop of ileum can rupture spontaneously and give rise to a localized walled-off abscess resembling a tumor on the pedicle. It is also interesting to note that the walled-off abscess continued to grow for six months before a new process resembling a volvulus took place, thus calling attention to its presence.

ZUSAMMENFASSUNG

Es wird über einen Fall berichtet, in dem nach Ansicht des Verfassers ein spontaner Durchbruch des Dünndarms mit Abszessbildung in ein Fettpolster des Ileums erfolgte. Der Darmriss fand offenbar beim ersten Anfall sechs Monate vor der Untersuchung statt. Während der Abszess im Fettpolster innerhalb der sechs Monate sich vergrösserte, legte sich das Omentum um den Abszess herum und lokalisierte auf diese Weise die Erkrankung. Ein weiteres Wachsen des Abszesses führte zu einer Drehung des Omentums um seinen Stiel und zu einer Drehung und Verschlingung einer Ileumschlinge.

Der Fall wird veröffentlicht, um auf einen seltenen Mechanismus aufmerksam zu machen, der zu einer spontanen Zerreissung einer Ileumschlinge und zur Entstehung eines lokalisierten abgekapselten einer Geschwulst des Stieles ähnelnden Abszesses führen kann. Es ist ferner von Interesse, dass in diesem Falle ein Wachstum des abgekapselten Abszesses sechs Monate lang vor sich ging, bis es zu einem neuen Krankheitsprozess kam, der dem Bilde einer Darmverschlingung ähnlich war und dadurch die Aufmerksamkeit auf das bestehende Leiden erweckte.

CONCLUSIONS

L'auteur présente un cas qu'il qualifie de rupture spontanée du grêle avec formation d'abcès dans un pannicule adipeux de l'iléon. La rupture s'est vraisemblablement produite six mois avant la seconde crise, qui a conduit le malade chez le médecin. Pendant cette période l'épiploon s'est enroulé autour d'un abcès augmentant de volume, localisant ainsi l'état pathologique. L'abcès, devenant encore plus volumineux, a provoqué un volvulus d'une anse iléale.

Ce cas attire l'attention sur un mécanisme rare de rupture spontanée d'une anse iléale accompagnée d'un abcès cloisonné, simulant une tumeur. Il est à noter aussi que dans ce cas l'abcès s'est développé pendant six mois avant de provoquer le processus décrit.

CONCLUSIONES

Se describe un caso en el que según la opinión del autor se trata de una rotura expontánea del intestino delgado con formación de abscesos en la capa grasa del íleon. La rotura ocurrió apartemente seis

meses antes de la observación en un primer ataque. A medida que los abscesos en la capa grasa fueron creciendo, durante un período de 6 mese, el epiplón fue envolviéndose en torno al íleon localizando así el proceso. Cuando el absceso se hizo mayor aún se retorció sobre su pedículo originando un vólvulo del asa del íleon.

Este caso es descrito con el fin de llamar la atención sobre este raro mecanismo por el cual un asa del intestino del gado puede romperse expontaneamente y dar lugar a abscesos localizados y aislados que recuerdan tumores pediculados. Es tambien interesante hacer notar que en este caso los abscesos encapsulados continuaron creciendo durante 6 mese hasta que se presentó una nueva manifestación semejando un vólvulo, y llamó atencion sobre su presencia.

CONCLUSAO

Um caso em que na opinião do autor, observou-se ruptura espontânea do intestino delgado seguido deformação de um abcesso em um apêndice gorduroso do ileum. O ruptura aporentemente tinha ocorrido seis mêses antes do exame fisico durante o primeiro ataque. A medida que o abcesso crescia no período de seis mêses o epiplon envolven o processo localizando-o. O crescimento posterior do abcesso ocasionou torsão e vôlvulo de uma alça de intestino delgado. Este caso é apresentado para chamar a atenção para um mecarismo raro no qual um segmento de ileum pode sofrer ruptura espontânea, dando origem a um abcesso localizado que pode assemelhar-se a um tumor com pediculo.

Outro aspecto de interêsse é que no caso presente o abcesso continuou a crescer durante três mêses até ocasionar o vôlvulo, chamando então atenção para a sua presença.

CONCLUSIONI

Viene presentato un caso di rottura del tenue probabilmente spontanea, con formazione di un ascesso. La rottura si era prodotta circa sei mesi prima dell'esame, e poichè l'ascesso era cresciuto di volume, l'omento lo aveva circondato localizzandolo. L'ulteriore sviluppo della sacca ascessuale aveva determinato la sua torsione sul peduncolo e quindi il volvolo dell'ansa di ileo.

Il caso richiama l'attenzione sulla possibilità di rottura spontanea di un'ansa ileale, con un raro meccanismo, e con la produzione di un ascesso pseudoneoplastico ben localizzato nel grasso.

E' interessante anche il lento sviluppo dell'ascesso, fino alla produzione di un volvolo che richiamò l'attenzione sulla lesione.

The last of the great prehistoric inventions was the art of writing, which was indeed a prerequisite of history. Writing, like speech, developed gradually, and in the form of pictures designed to convey a message it was probably as old as speech, but from pictures to syllable writing and thence to the alphabet was a very slow evolution.

—*Russell*

Transcystoscopic Injection of Tumors of the Bladder with Radioactive Colloidal Chromic Phosphate

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CARCINOMA of the bladder, which is inoperable by virtue of its position or the condition of the patient, presents a therapeutic problem that is difficult and often impossible to meet successfully.

Irradiation has, in some instances, proved to be a satisfactory controlling agent for such malignant tumors,[1] and there is good evidence of the curative value of radon seeds. Such success has led to speculation and actual investigation of the possible value of certain radioactive isotopes when injected interstitially.[2] Colloidal suspensions of such materials can be handled by transcystoscopic injection. The ease of this approach is striking, since it produces a minimal reaction and allows greater access to some of the more difficult tumor sites. This paper details an attempt to develop such a technic and to perfect an approach that is simple and safe for both the patient and the hospital personnel. In so doing, animal experimentation was first employed. Later, our observations in the laboratory were taken directly to the clinical patient.

Material and Method.—Four large female dogs were used as experimental animals in the first group. With the dog under pentobarbital sodium anesthesia, a No. 16 panendoscope was introduced into the urinary bladder. A dilute solution of colloidal carbon buffered to a pH of 7.5 was then injected via a No. 6 French ureteral catheter needle into an area 2 cm. in diameter on the posterior aspect of the bladder, as follows: 0.5 ml. at 0.5 cm. intervals, 0.5 ml. at 1 cm. intervals and 1 ml. at 1 cm. intervals.

Forty-eight hours after the injections the animals were again anesthetized with intravenous pentobarbital sodium, and the bladder was removed, opened longitudinally and inspected. Sagittal sections were made through the areas of injection, and observations were made with regard to the confluence of the colloidal carbon particles. Specimens were then taken for microscopic section.

The animals of the second group were anesthetized and treated in the same manner except that P^{32}, in the form of colloidal chromic phosphate, was used instead of the colloidal carbon suspension. The concentration of this solution was 50 microcuries per milliliter; thus, in a 2 cm. area

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in the posterior wall of the bladder, a total dose of 500 to 700 microcuries was used. Twenty-four hours later one-half of the animals in this group were again anesthetized; a cystectomy was done as aforedescribed and samples taken for radioautographic study. The remaining animals were held for forty-eight hours, after which the same procedure was then carried out.

Results.—In the first group, i.e., the dogs given carbon injections, the most desirable distribution was achieved with the injection of 0.5 ml. at 0.5 cm. intervals (Fig. 1). Slightly less desirable results were obtained by the use of 1 ml. at 1 cm. intervals. It was observed that moderate edema was present, with minimal extension of the carbon beyond the serosa of the bladder wall so as to involve the extravesical adipose and fibrous tissue in an area slightly larger than the site of injection into the muscularis and submucosa. Grossly, the confluence of particles was uniform and continuous throughout the muscularis (Fig. 2). This was interesting, as the difficulty of accurate measurement of small distances in an organ as contractile and movable as the bladder is known to all who have employed transcystoscopic technics.

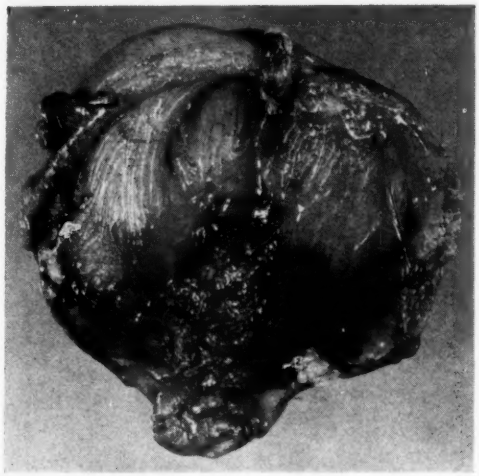

Fig. 1.—Specimen from dog given carbon injection (0.5 ml. at 0.5 cm.). This resulted in the best distribution of the injected material (see text).

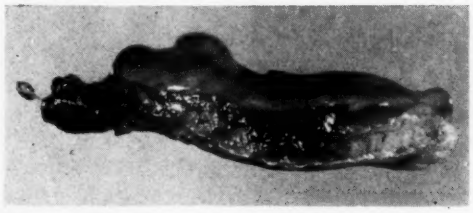

Fig. 2.—Slightly less desirable distribution obtained by carbon injection of 1 ml. at 1 cm. (see text). Note minimal extension of carbon beyond the serosa of the vesical wall.

The radioautographs showed that the radioactive colloidal chromic phosphate behaved interstitially in a manner parallel to that of the colloidal carbon. Here, intervals of 0.5 cm. and volumes of 0.5 ml. containing 25 microcuries of radioactive chromic phosphate gave the most desirable distribution of radiation, but a uniform spread was also obtained by injecting 1 ml. at intervals of 0.5 cm. When the distance was increased to 1 cm., however, the areas showed definite patchy distribution. Radioautographs taken twenty-four hours after injection showed strict localization, which became confluent after forty-eight and seventy-two hours (Fig. 3 and 4). It was also noted that there was practically no escape into the perivesical tissues. A quantitative assay showed nearly complete retention of the isotope in the bladder wall within sixty hours.

Technic and Injection.—The major clinical problems required the development of a technic that would be safe for the patient who was unable to withstand an extensive operative procedure and also feasible in those cases in which the malignant growth was not accessible for local excision. The technic, it was realized, would also have to be clinically applicable by any competent urologist.

In nearly all cases a low spinal anes-

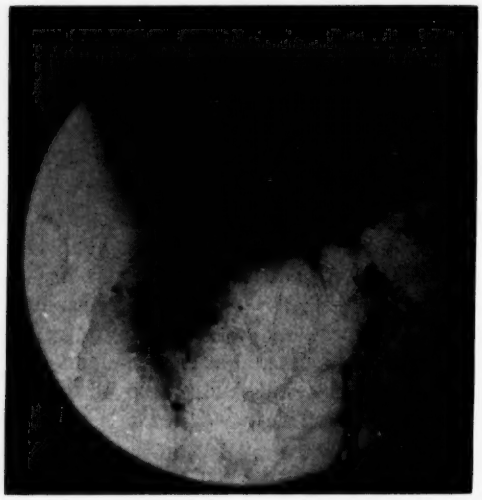

Fig. 3.—Radioautograph showing strict localization of injected radioactive chromic phosphate (see text).

thesia of short duration was used. This entailed a minimum of anesthetic hazard to the patient and eliminated as far as possible the danger of explosive intravesical pressure, which could cause sudden escape and contamination.

The equipment for injection comprised the following items: a No. 24 Brown Burger cystoscope, a single carrying operative lens, and a No. 6 French ureteral catheter, to one end of which a ¼ inch (0.6 cm.) 25 gauge needle was attached and to the other a regular luer-lok adaptor (Fig. 5). For protection of the operating surgeon, a polyethylene sheet was gathered to fit lightly over the head, forming a complete, waterproof hood. Plain glass spectacles were glued to holes cut in the plastic (Fig. 6). The plastic opposite the observing eye was removed to prevent fogging. Two pairs of rubber gloves were worn to provide protection. Nothing else varied from the ordinary cystoscopic technic. Plain glass spectacles were also worn by the injector in case of leakage due to faulty connections.

A separate sterile table was used for segregating all radioactive solutions and contaminated instruments. Two ml. of sterile indigo carmine or methylene blue was placed in the chromic phosphate for visual tagging of the isotope solution.

The lesion, which had been previously appraised and biopsied, was again observed. The injector, standing back of the

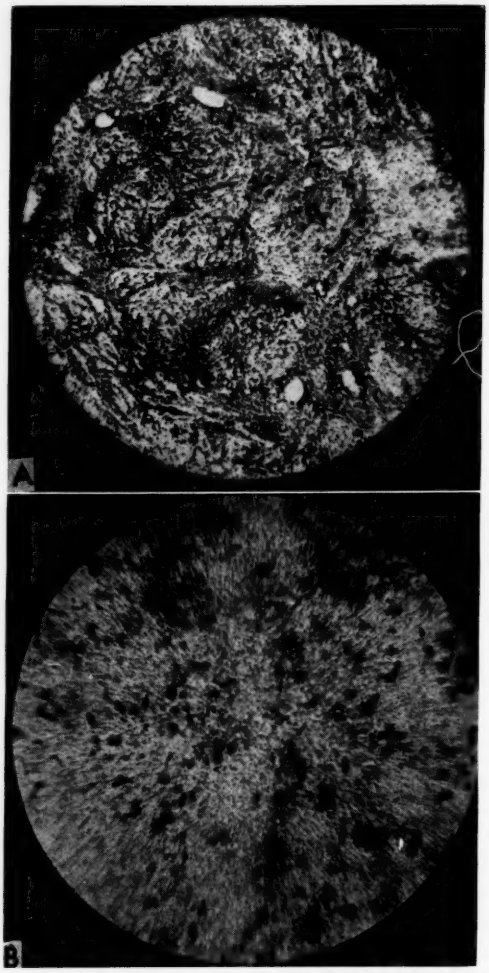

Fig. 4.—*A* and *B*, radioautographs showing confluence of radioactive chromic phosphate after twenty-four hours.

operating surgeon, slowly delivered the isotope solution through the catheter until the appearance of the blue stream indicated readiness to implant. With the needle inserted, 0.5 ml. or any increment thereof could be injected at the command of the surgeon. A minimum of irrigating fluid was used, and the bladder generally was not emptied more than once or twice during the injection. Great care was taken to prevent spillage on the table or the personnel, even though the dilution was great. After completion of the injection, the bladder was finally filled through the cystoscope and then emptied into an enamel receptacle. This wash water was poured into an enamel sink in which water was running. All equipment was immediately placed upon the isotope table. The patient was then monitored and returned to his room with no further precautions.

The cystoscopy table was then monitored, and if activity over 2 milliroentgens per hour was observed, the contaminated areas were scrubbed with Phisoderm until the activity dropped below this level. The cystoscope was then monitored and washed, although up to the time of writing, practically no contamination has been detected. All other linen and equipment, together with the hoods, gloves, gowns and aprons of the injector and the surgeon, were individually monitored and if any activity in excess of 2 milliroentgens per hour was detectable, the article was placed in a sealed box and stored in an isolated closet for twenty-one days or until the emanations had dropped below 2 milliroentgens per hour.

Immediately after the operation the patient was returned to bed. Any distress was usually controlled with codeine. Only mild suprapubic discomfort was recorded in most instances. The patient was usually discharged after twenty-four hours' hospitalization and a check cystoscopic examination was done in six weeks. With

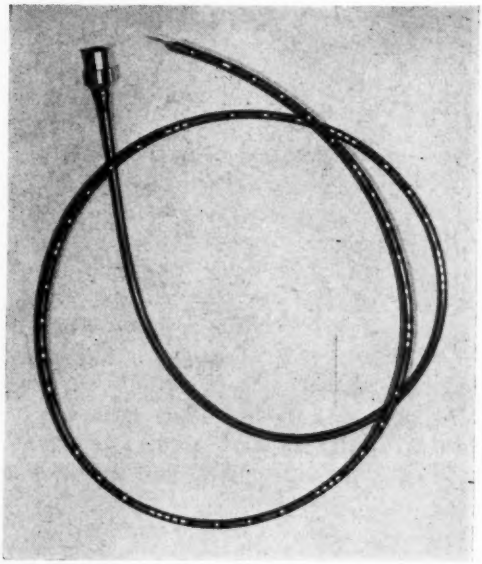

Fig. 5.—French ureteral catheter (No. 6) employed, with ¼ inch 25-gauge needle attached to one end and regular luer-lock adapter to the other.

the concentrations of the isotope used thus far we have encountered no vesical or urethral fistulas.

Clinical Data.—Previous treatment of vesical carcinoma by interstitial injection has been reported.[2a] The procedure appears to have real merit. The series of patients treated thus far is certainly no more than a pilot series, and the individual cases have been selected either on the basis of inability to survive operation or because the location of the tumor was such as to make local or radical excision impractical. Furthermore, most of the patients had been treated unsuccessfully by other methods.

Six cases were selected because of a Grade III or IV papillary carcinoma incorporating either one or both ureteral orifices and showing mild to moderate ureteral obstruction on the intravenous program. Chromic phosphate was injected through and around the ureteral orifice in an amount considered sufficient

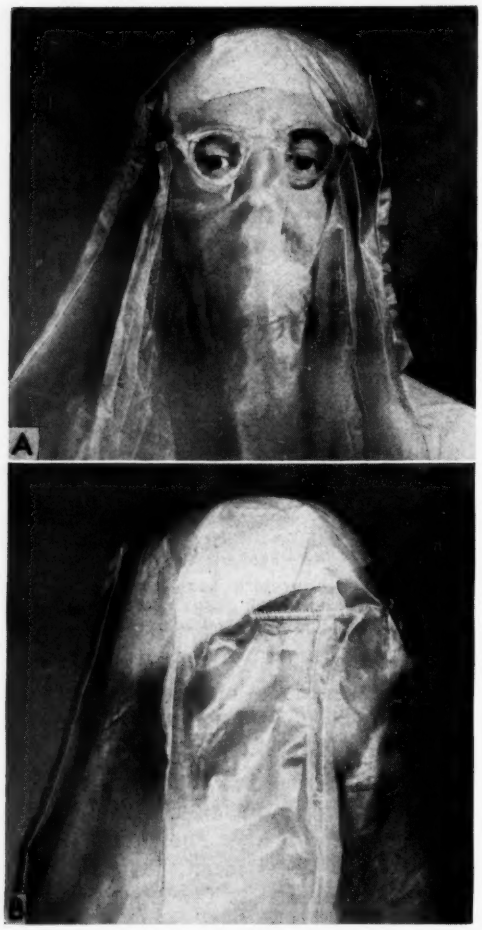

Fig. 6.—Frontal (A) and lateral (B) views of waterproof polyethylene hood used for protection of the operating surgeon.

to eradicate the tumor, but thus far no stricture formation has resulted. Hydronephrosis has subsided and a normal configuration of the intrarenal structures has returned in two-thirds of the cases involved. In these patients, however, only one-third to one-half of the total tumor area was treated by injection. In approximately 60 per cent of the cases there was marked hematuria, beginning three weeks after the injection and lasting for two to three weeks. Since these tumors had been only partially treated, it is possible that the hematuria was coming from the sites into which no injection had been made. Six to eight weeks after the injection, tumor was no longer apparent in the areas of injection. The mucosa showed moderate telangiectasia; pyuria was absent in most cases.

COMMENT

From both the laboratory and the clinical data we concluded that an additional adjunct to the therapy of carcinoma of the bladder is possible for patients who may be otherwise considered inoperable. Although no definite conclusions should be drawn from a pilot series, the disappearance of lesions, particularly in patients who had been treated by at least one other method, gives sufficient validity to the transcystoscopic injection to justify further treatment and study.

The first injection was done with gold 198. Thereafter we changed to chromic phosphate, for much the same reasons mentioned by other authors. Since the P^{32} has a half-life of fourteen days, there is a reasonable latitude in time for receiving shipments and performing injections. Decontamination has not been a serious problem. Since P^{32} is a pure beta emitter, it offers safer handling with less exposure to personnel than do isotopes with gamma emission. Because of the short penetrating distance of beta radiation, the patients treated therewith present no additional nursing problems. All safety precautions in the handling of the isotope are aimed primarily at keeping it off any exposed cutaneous surface. It is extremely tenacious and requires immediate, vigorous scrubbing to achieve adequate removal.

It is quite conceivable that this technic could be generally applied by competent urologists and not confined to teaching centers as a clinical curiosity. Thus far we have had a low morbidity rate, and hospitalization has been minimal. As far

as can be determined, the procedure requires little more convalescence than does a cystoscopic examination procedure. Repeated injections have been done at intervals of thirty to sixty days, accompanied by a cautious check of the blood picture. It has been observed by others that from 4 to 5 per cent of the injected isotope is absorbed into the blood stream, but when a total dose of 0.5 millicuries is used, 4 to 5 per cent absorbed systemically should not cause appreciable changes.

The transcystoscopic approach can be used successfully in any location where a needle can be introduced, but especially around the vesical neck, the trigone or the posterior portion of the urethra in the male. We suspect that it will be best to confine the injection to tumors with a diameter of less than 3 cm. or to multiple small ones.

SUMMARY

In cases of inoperable carcinoma of the bladder, the authors suggest the use of interstitial transcystoscopic injection of radioactive colloidal chromic phosphate. On the basis of animal experimentation and clinical practice they are convinced that this method has great advantages in the treatment of lesions in locations difficult of access; in addition to this, the reaction is minimal. Using dogs, the authors satisfied themselves of its efficacy by experiment, later taking the laboratory observations directly to the clinical patient.

The technic and equipment used are described in detail. Six clinical cases were selected, in all of which Grade III or IV-papillary carcinoma was present. Chromic phosphate was injected around the ureteral orifice in an amount considered sufficient to eradicate the tumor. No stricture has resulted; hydronephrosis has subsided, and return of the intrarenal structures to their normal configurations has occurred in two-thirds of the cases. In these patients, however, only one-third to one-half of the tumor area was treated by injection.

The authors emphasize the fact that no definite conclusions should be drawn from a pilot series, but express the opinion that this procedure may well be a valuable adjunct to the treatment of vesical carcinoma. Radioactive chromic phosphate (P^{32}) emits pure beta rays and is therefore safer to handle, with less exposure of hospital personnel, than are isotopes with gamma emission.

The transcystoscopic approach can be used successfully wherever a needle can be introduced but is especially well adapted to the vesical neck, the trigone or the posterior portion of the male urethra. It is the authors' present impression that the injections are best confined to small multiple tumors or to single ones under 3 cm. in diameter.

RESUMEN

Los autores aconsejan la inyección intersticial transcistostópica de fosfato crómico coloidal radioactivo en los casos de carcinoma inoperable de la vejiga. Existen datos suficientes clínicos y de laboratorio para demostrar le eficacia de este método, justificando neuvos estudios y posiblemente un mayor uso.

Según la opinion del autor este método disminuye ampliamente el peligro asociado con el empleo de isótopos radiactivos tanto para el enfermo como para el personal del hospital. El fosfato crómico coloidal (P^{12}) es un simple beta-emisor, y puede ser manejado con la máxima seguridad, ya que la corta distancia de penetración de los rayos beta no plantea problemas especiales en el personal auxiliar. El abordaje transcistoscópico se puede usar en toda localización en la que pueda ser introducida la aguja.

RIASSUNTO

Per la cura dei carcinomi inoperabili della vesciça, gli Autori raccomandano l'iniezione di fosfato cromico colloidale radioattivo, da praticare per via transcistoscopica nelle pareti della vescica.

Dati clinici e di laboratorio dimostrano la validità di tale metodo terapeutico, tanto da giustificare ulteriori studied una più ampia applicazione. Secondo gli Autori, inoltre, il metodo diminuisce di molto il rischio connesso con l'uso degli isotopi radioattivi. Il fosfato cromico colloidale, per la sua composizione chimica e per le sue proprietà, può essere maneggiato con la massima tranquillità e non crea, perciò, altri problemi tecnici. La via transcistoscopica può essere usata in ogni sede dove possa essere introdotto un ago.

RÉSUMÉ

Dans les cas de carcinome inopérable de lavessie, les auteurs recommandent l'injection transcystoscopique interstitielle de phosphate de chrome colloïdal radioactif.

Les résultats cliniques et de laboratoire montrent que cette méthode mérite des recherches plus approfondies, et peut-être une plus large application. Elle diminue les dangers—et our le malade et pour le personnel hospitalier—de l'utilisation des isotopes radioactifs. Le phosphate de chrome colloïdal (P 12) est un émetteur de rayons Beta, et il peut être manié avec un maximum de sécurité. Du fait de sa faible pénétration, il permet d'éviter des soins supplémentaires. La voie transcystoscopique peut être utilisée pour toutes les localisation où l'introduction d'une aiguille est possible.

ZUSAMMENFASSUNG

Die Verfasser empfehlen zur Behandlung des inoperablen Blasenkrebses die durch ein Zystoskop erfolgende interstitielle Einspritzung von radioaktivem kolloidalem Chromphosphat. Sowohl die klinischen Beobachtungen als auch die Laboratoriumsbefunde ermutigen zu weiterer Untersuchung und veilleicht zu umfangreicherer Anwendung des Verfahrens. Die Verfasser glauben, dass ihre Methodedie mit dem Gebrauch radioaktiver Isotope einhergehenden Gefaren für den Patienten und das Krankenhauspersonal erheblich herabsetzt. Das kolloidale Chromphosphat (P^{32}) gibt ausschliesslich Betastrahlen ab und lässt sich mit einem Höchstmass an Sicherheit verwenden da die Betastrahlen nur eine goringe Eindringungsfähigkeit besitzen, entstehen keine das Pflegepersonal betreffenden Probleme. Der Zugang durch das Zystoskop ist in allen Lokalisationen, wo eine Nadal eingeführt werden kann, möglich.

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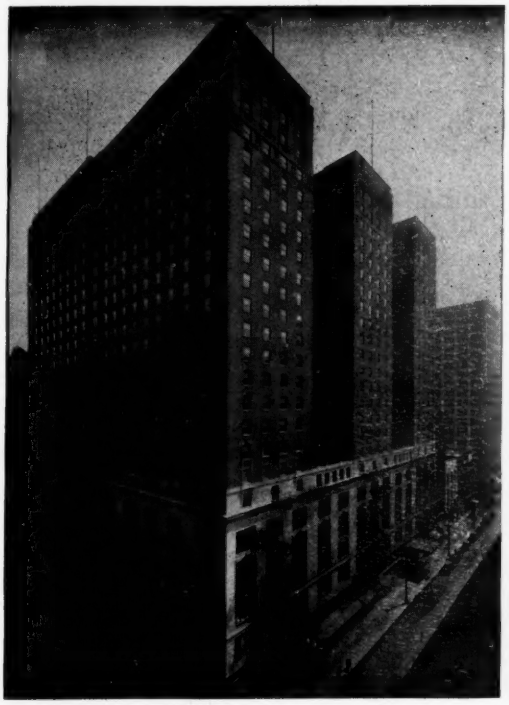
United States and Canadian Sections

International College of Surgeons to Hold Twenty-Second Annual Congress in Chicago Sept. 8-12, 1957

Members and friends of the International College of Surgeons are cordially invited to attend the Twenty-Second Annual Congress of the United States and Canadian Sections of the International College of Surgeons, to be held at the Palmer House in Chicago, Sept. 8-12, 1957. A timely and stimulating scientific program, including the ever-popular Film Forum, has been arranged, and an impressive array of up-to-the-minute exhibits (see pp. xxiii-xxviii inclusive) will be on display. In addition, special social events and entertainment will be offered to the visiting surgeons and their wives. An application blank for hotel reservations will be found on p. xxxiii.



The Palmer House, Chicago, where the Twenty-Second Annual Congress of the United States and Canadian Sections of the International College of Surgeons will be held Sept. 8-12, 1957

Neurologic Surgery

Low Back Pain and Sciatica Due to Posterior Compression

STEFANO TENEFF, F.I.C.S.

TURIN, ITALY

IN recent years there has been a renewal of studies attempting to define minutely the pathogenesis and treatment of sciatica and pain low in the back. It is not my purpose to record in this article the history of the research evolution that led to the present level of competence in determining the cause of such pain and providing adequate treatment to assure recovery. It is well to call to mind, however, that although isolated observations of radicular compression, especially in cases of disc protrusion, have been known since Luschka, in 1858, demonstrated by autopsy the existence of hernia, it is actually since the Second World War that it has been perceived that sciatica is a disease not of the sciatic nerve and the roots that compose it but of the spine. The pain was attributed to causes which, though they have great pathologic importance in relation to the causes discovered later, still were not the actual factors that produce the syndrome. The importance of slight inflammatory processes must be remembered, as must the dilation of the blood vessels that surround the roots of a compressed sciatic nerve, called by Sicard neurodocite; of the diminution in amplitude of the intervertebral foramens, and of occasional signs such as alteration of the adipose tissue of the intervertebral foramens or tumors of different kinds in that area. Kocher first described, in 1896, a clinical case of protrusion of the disc. Oppenheim and Krause were the first to describe a case in which the treatment was surgical. It must be admitted, however, that Alajouanine and Petit Dutaillis were the first (1930) to establish the fact that in numerous cases radicular compression is to be attributed to posterior hernia of the disc towards the vertebral canal, as described by Schmorl. It was only in 1940 that Love and Walsh published statistics on 500 operative cases. In 1943 Mixter, Barr and Hampton described 300. Since then statistics have multiplied, and contributions on the subject have been presented at many congresses, as well as in the literature.

All these studies brought to view a fundamental fact, that sciatica and pain low in the back in most cases are to be attributed to radicular compression or of a mechanical irritation, because of the direct relation of the root to pathologic manifestations, reversible and irreversible, at the level of the vertebral canal and the intervertebral foramens.

Although until a few years ago the syndrome of compression and irritation of the roots was rarely attributed to protrusion of a disc, there is now a tendency on the

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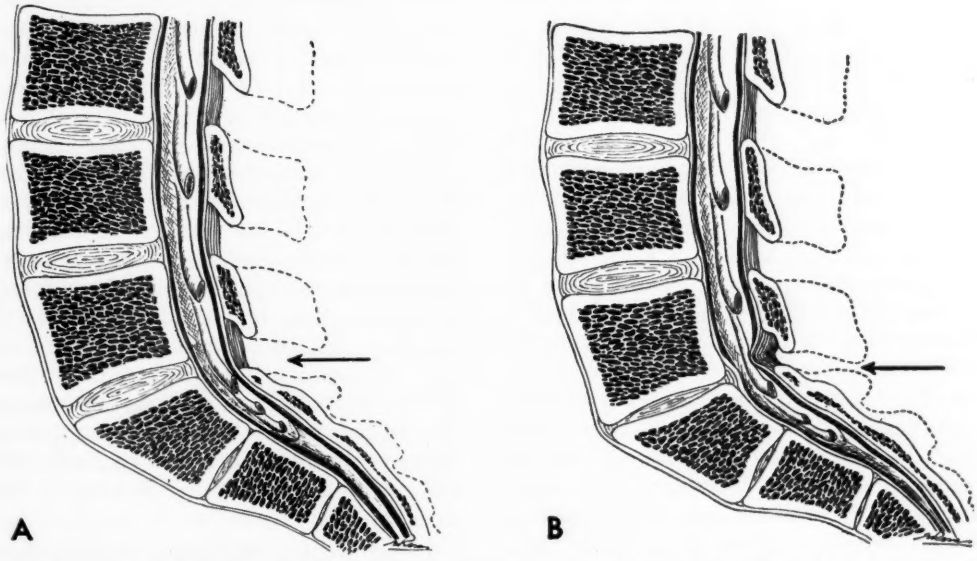
part of some surgeons to attribute the syndrome in every case to the presence of a protruded disc. These surgeons affirm that when the clinical diagnosis of radicular compression and irritation is made with the help of clinical examination and of various diagnostic procedures (myelographic and discographic studies, tests of the spinal fluid, peripheral electric tests), the protruded disc must always be present. This tendency is already evident in the titles of their publications, e.g., "Intervertebral Disk Syndrome." On accurate analysis of the observations it is noted that in a certain percentage of cases the presence of a protruded disc was not comfirmed at surgical intervention or its absence was obvious, although aside from the details of direct observation, every diagnosis of cause stressed the syndrome of sciatic pain.

Regarding this fact we refer to some of the most recent statistics. In 1954, of 1,104 operative cases of sciatica reported by Scaglietti with a diagnosis of disc syndrome, hernia was encountered in 1,020; therefore, in 84 cases (7.61 per cent) radicular compression, confirmed by all diagnostic means available, was not due to protruded disc. On 1,000 operative cases reported by Pais and Picchio, in 20 cases it was a question of rebel pain low in the back; in 906 the sciatica was of discal origin, and in 94 (9.4 per cent) there was no hernia of the disc, but other conditions capable of causing radicular irritation, and compression were present. Of 114 operative cases of sciatica reported by Padula and Keys, hernia of a disc was present in 106, and in the remaining 8 cases the pain was attributed to the varicose dilations of the anterior lateral plexus of the dura-mater to arthritic manifestations with hyperproduction of bone. On



A, schematic representation of the yellow ligaments in normal conditions. The lumbar and sacral spine dissected longitudinally according to lateral plane. *B*, schematic representation of the anterior protrusion of the yellow ligament between the fifth lumbar vertebra and sacrum in the degeneration of disc and sinking to the fifth lumbar vertebra with the posterior arch. Posterior compression on the first sacral root. The lumbar and scaral spine dissected longitudinally according to lateral plane.

61 operative cases of sciatica reported by Bonola a protruded disc was present in 53; so-called hypertrophy of the yellow ligament in 3; tumors of various kinds in 3, and periduritis with compression of the first sacral root in 2. In a later publication by the same author reporting 220 operative cases of sciatica, the pain in 3 proved to be the result of congenital anomalies of the spine and of radicular emergency, which Ethelber and Rishede seem to have observed to be the only cause of sciatica in their series, while Fineschie and Plandri observed it in association with hernia of the disc. Krayenbühl and Zander operated in 1,700 cases of sciatica and low back pain with the diagnosis of disc protrusion, in 2.4 per cent the operation failed to reveal a protruded intervertebral disc but did reveal spondylolisthesis, vertebral tumors or spondylitis; in some cases it was not possible to ascertain the pathogenesis of the radicular compression and irritation. Numerous other publications contain similar statistics.

It is well known that, apart from hernia of the intervertebral disc, the most common conditions that may cause low back and sciatic pain are degeneration of the intervertebral disc of Putti, arthritic apophysitis of Putti, arachnoiditis, periradiculitis, periduritis, lumbar scoliosis, congenital or acquired anomalies, lumbar intersomatic deforming arthrosis with osteophytic proliferations, so-called hypertrophy of the yellow ligaments, spondylolisthesis, sacroileitis (most frequently of tubercular origin), peridural and periradicular varicose veins, apophyseal fractures, fractures of the margins of the vertebral bodies and tumors of various types. Any of these may be isolated or associated with hernia of a disc. In one statistical report based on cases of inveterate pain that had persisted for many years, the percentage was high. On clinical examination based on one's judgment of the results of subsidiary diagnostic research in cases of disc hernia associated with other pathologic conditions, especially deforming arthrosis, with degeneration of the intervertebral disc, arthritic apophysitis, scoliosis and traumatic conditions, it is often impossible to conclude with certainty whether the sciatalgic syndrome (irritation and radicular compression) is due to the protruding disc, to the associated conditions or to both, since the clinical symptoms are not substantially different. The "disc syndrome" may appear with all its manifections not only in a case of hernia of the disc, but association with any other of the conditions mentioned. In a considerable percentage of the cases, except for the characteristic constant pain, only a few of the characteristic symptoms can be verified; they may even be absent when the condition is of a type that is slow to cause symptoms that are considered important.

Sometimes even those fundamental signs are missing which should help in the clinical diagnosis of hernia of a disc. In a certain number of cases, patients who have been complaining for several years of low back and sciatic pain, objective clinical symptoms are entirely absent. Faced with such varying behavior, one is sometimes perplexed about the therapeutic decision, but even in this field, despite the remarkable growth of experience and knowledge, it rests on intuition, as in other fields of surgery. It is not possible, therefore, to base the decision on subsidiary researches (discographic and myelographic study), for neither the positive nor the negative evidence of the reports can be pathologically or anatomically decisive as to the cause of sciatic pain.

In my opinion, therefore, what matters in cases of low back pain and sciatica, except in the presence of chronic inflammatory diseases of the spine, such as tubercular spondylitis, it is not the preoperative

anatomic-pathologic diagnosis that counts, but a precise diagnosis showing that in correspondence to the lumbar and sacral roots there exists a cause of irritation and compression surgically mastered. If preoperative tests have established exact cause of this radicular compression, so much the better, and of course all diagnostic efforts are directed to this. Even when doubts exist, as to whether the therapeutic indications are the same in the two eventualities, interoperative diagnosis will be better able to establish the anatomic-pathologic origin of the pain and provide treatment accordingly. It must be borne in mind, at the beginning of every intervention for low back pain and sciatica, that it may not be a question of hernia of the disc and also that the hernia of the disc, even if present, may not be the only cause of the painful syndrome; otherwise serious mistakes may be made during interoperative diagnosis and in deciding on the necessary technical management. It is only by following these directions that it is possible to insure the complete recovery of the patient.

During preoperative and interoperative analysis of all these facts it is important to remember, so as to avoid serious omissions, the direction from which irritation and compression of the roots may arise. Given the anatomic shape of the vertebral canal and bearing in mind the topographic relations of the roots in their interrachidian passage and through the intervertebral foramens, the pathologic irritation and compression may be manifested in the frontal, lateral and posterior positions. With hernia of the disc and other pathologic processes referable to the disc, the cause of pain is anterior to the root. In cases of interapophyseal arthrosis and osteophytosis, which are caused by articular pathologic processes, and in some cases of ligamental ectopic proliferation, the cause of the pain is lateral to the roots.

On the basis of about 500 cases I have been able to ascertain that in a remarkable percentage the cause of radicular irritation and compression is posterior to the site of the root involved. In most cases the compression is only anterior; in a certain number of cases it is simultaneously anterior and posterior. There are cases, however, in which the roots are compressed by anterior, posterior and sometimes also lateral compression, and the space they must pass through is only that of the volume of the root.

It has seemed necessary to emphasize the importance of posterior compressions, trying to fix and define them with accuracy, because during laminectomy one may eliminate them without arriving at a precise interoperative diagnosis of the cause of low back pain and sciatica, which makes the success of surgical intervention doubtful. They must also be known, because they must be examined in every case of intervention, so as to avoid every possibility of return of the pain even when they have been obvious and when the more frequent causes of the pain have been eliminated, especially those associated with hernia of the disc. These causes of posterior compression may be: (1) pathologic manifestations in the yellow ligaments; (2) increase of the thickness of the laminae; (3) congenital or acquired anomalies (a) excessive protruding in front of the superior margin of the sacral lamina; (b) sinking toward the front of the posterior arch, especially of the fifth lumbar vertebra, with lordosis, and accentuated when associated with horizontalization of the sacrum and with spondylolisthesis; (c) lumbar scoliosis, which may present many varieties of gradation and anatomic-pathologic presentation, and (4) tumors of the arches or tumors invading the posterior aspect of the intervertebral space.

In all these circumstances the space re-

served to the roots, corresponding to the level at which the anatomic-pathologic modifications are manifest, is reduced in the posterior-anterior direction, and the roots are irritated or compressed in the same direction. While all the other aforementioned circumstances need no particular description and their action on the root can be perceived by intuition, I wish to lay particular stress on pathologic changes in the yellow ligament and radicular compressions referable to the superior margin of the sacral arch. I have chosen a more generic term on purpose: "pathologic changes in the yellow ligaments" instead of "hypertrophy of the yellow ligaments," although the latter has entered into the common nomenclature. The first case was described in 1913 by Elsberg. Successively numerous other cases have been described in the literature of every country; so many, in fact, that in the most recent publications the statistics of frequency are included in the reports. This anatomic-pathologic picture may represent the only cause of this painful syndrome, according to Love, in 4 per cent of the cases; according to Pais, in 2 per cent. More frequently it is associated with hernia of the disc; according to Love, in 6.86 per cent; according to Spurling and Bradford in 8.33 per cent; according to Delitala and Bonola, in 5.1 per cent, and according to Pais and Picchio in 15 per cent.

The cause of radicular irritation and compression seems to be an increase in thickness of the yellow ligaments, due to degeneration and inflammation caused by traumatic lesions or professional microtraumatic lesions that have caused lacerations of the collagen and elastic fibers of the yellow ligaments, hematoma formation and subsequent cicatrization. Although normally the yellow ligaments are 3 to 3.5 mm. thick, in these circumstances their thickness reaches 10 to 20 mm. The yellow ligaments, like all dense ligamental formations, especially when they are rich in elastic fibers, are subject to degenerative cicatricial and sclerotic alterations. I have often observed these alterations even when there was no increase in thickness, nor was it a question of posterior root compression, especially when there was abnormal mobility of the vertebral column (which will be the object of another study). It is urgent to ascertain that, although, bearing in mind the exact description of the cases reported in the literature, it seems to us, that it has not been clearly proved that a real increase of the thickness of the yellow ligament has occurred to provoke radicular irritation with consequent low back pain and sciatica.

I observed this anatomic-pathologic picture in 10 cases, in 4 of which it was the only cause of posterior radicular compression with pain. In 6 it was associated with posterior protrusion of the disc at the same level, always between the fifth lumbar vertebra and the sacrum.

An accurate preoperative test, especially the taking of anterior-posterior and laterolateral roentgenograms and accurate and minute interoperative observations during partial laminectomy, have persuaded me that the increase in the thickness of the yellow ligaments is to be considered not real but fictitious. In reality the anterior-posterior diameter of this ligament is met with greatly increased pressure, and it protrudes more or less toward the vertebral canal, while at the same time the distance between the insertions on the laminae at the same height are diminished. In all the cases I have studied these alterations in the yellow ligament have been accompanied by a diminution at the level of the space between the vertebral bodies, caused by degeneration of the disc or by the presence of a posterior hernia; in the second instance the diminution of the disc volume sometimes is only partial and in correspondence with its

posterior aspect.

In total or partial reduction of the thickness of the intervertebral disc, the vertebra above it, with its body and its posterior arch, approaches the corresponding anatomic parts of the vertebra below it. The laminae of the vertebra above it protrude toward the lower part, and their lower margin approaches the superior margin of the laminae of the vertebra below it. On account of the anatomic configuration of the laminae the respective margins never touch, because those of the upper vertebra slip downward and backward, crossing over those of the vertebra below it. The space intended for the yellow ligament, which is between these two laminae, diminishes in height, sometimes one-third, sometimes about one-half. As the laminae are composed of hard, elastic tissue, they are not crushed, but as long as they find space they bend toward the vertebral canal, where there is no resistance—never in the posterior direction, being hindered by the lower margin of the lamina of the vertebra above. The forward protrusion of the yellow ligament is additionally determined by the form of the site of insertion above. This behavior of the yellow ligament is represented in Figures 1 and 2.

During the destruction of the arches and of the yellow ligaments for the performance of laminectomy, it is evident that the thickness of the yellow ligaments has increased, but this increase, as aforementioned, is due exclusively to the closeness of the stitches. The histologic observation of these anatomic-pathologic alterations in the yellow ligament is not surprising, for they are consequences of the same pathologic process that affects the lumbar space involved. This is also the case with the frequently reported pseudo-increase in volume of the yellow ligament in cases of hernia of the disc, and this too is not surprising, because hernia of the disc is one of the causes of the diminished space between the vertebrae and therefore of the bearing of the posterior arch of the lower vertebra to the vertebra underneath. These interpretations give more value to the considerations of Pais, who in 1946 affirmed that an increase in volume of the yellow ligaments and hernia of the disc are often pathologically interdependent. When associated with hernia of the disc, this formation constrains the root from before to behind such a protrusion, which, on the contrary, protrudes from behind to before.

Bilateral posterior compression causes alterations in the yellow ligaments, which may be more or less symmetric and more or less obvious. I have explained 2 cases of bilateral sciatica occurring simultaneously or intermittently in cases of diminution of the space between the vertebral bodies, with a typical disc hernia on one side only. The two patients recovered as a result of partial bilateral laminectomy with demolition of the spinal process and removal of the yellow ligament and the unilateral disc hernia.

Another important cause of posterior compression is *excessive forward protrusion of the upper margin of the sacral laminae.* The frequency of congenital anomalies of the tract, which comprises the fifth lumbar area and the sacrum, is well known. These malformations may be very evident, ranging from the sacralization of the fifth lumbar vertebra and from the lumbarization of the first sacral vertebra, in varying degrees, to the rachiskisis and other less evident malformations that cause protrusion of the upper margin of the sacral laminae against the disc between the fifth lumbar and the first sacral vertebrae. Between these the lumbar protrusion of the extreme margin of the sacrum can be observed in some cases of sciatica. It must also be mentioned that in some cases an excessive protrusion may

be the only cause of compression of the first sacral root, especially when, in the fourth or fifth decade of life, the horizontalization of the sacrum becomes more accentuated as a congenital anomaly. I have not observed such a case. Instead, this protrusion has assumed great importance in cases of hernia of the disc between the fifth lumbar vertebra and the sacrum. In 3 cases a small lateral hernia greatly compressed the first sacral root against the margin of the lamina of the sacrum, and it was flattened and herniated. It was therefore necessary to remove the hernia and also to demolish, as thoroughly as possible, the sacral lamina toward the bottom, in order to create a space sufficiently large for the involved root. Without this circumspection, it is presumable that any cause that may intervene in the postoperative period or later may contribute to reduction of the space through which the first sacral root passes. Without demolition of the sacral lamina, the possibilities of relapse are considerable.

From the interoperative observation of abnormal anatomic formations or alterations of the kind aforementioned, which may cause sciatica by lateral or posterior compression, it may be inferred that, in treatment of this painful syndrome by laminectomy according to the preferred method, it is not sufficient to eliminate the most obvious cause, which is generally hernia of the disc; it is necessary to remove all other causes that may have contributed to the irritation and compression of the root involved and may still cause a recurrence of pain, since they furnish the starting point of cicatrization and this in its turn may cause radicular compression and, consequently, low back pain and sciatica. In cases of hernia of the disc I proceed, after correction of this, to removal of the ligamental and fibrous formations, which are encountered toward the middle, near the respective interapophyseal articulations. In most cases these are threads of the yellow ligaments, which sometimes have a considerable thickness and are formed astride the respective roots. In some patients it is necessary to demolish one of the articular processes also. When the cause of compression is discovered at the level of the fifth lumbar vertebra and of the sacrum, I proceed to systematic removal of the protruding margin of the sacral laminae. In any case I perform a sufficiently large laminectomy, no matter what technic is used, according to the indications for intervention and the requirements of accuracy, and as far as possible I cut away the laminae at the level of the compression, as well as all the fibrous formations that may cause a posterior compression, especially of the yellow ligaments. By means of this procedure and having recourse to osteosynthesis according to Scuderi and Khedroo, in cases of instability of the lumbar portion of the spine and increased mobility of the fourth and fifth lumbar vertebrae as observed during the operation, and in cases in which the clinical and the roentgen tests have shown evidence of arthrosis of the small interapophyseal articulations, spondyloarthrosis, degeneration of the intervertebral disc with diminution of one or more of the intervertebral spaces, horizontalization of the sacrum, congenital anomalies of various kinds and other pathologic changes, I have obtained definitive recovery of the patient and have had no complaints of relapse.

Finally, in cases of persistent low back pain and sciatica, in addition to conservative treatment, all tests must be directed toward a precise diagnosis of the cause or causes. This must go beyond the clear diagnosis of hernia of the disc; it is necessary to look for the eventual presence of other causes of irritation and radicular compression as aforementioned. After the indications for surgical treatment have

been decided upon it is necessary to explore, with great accuracy, all the abnormal or pathologic formations that may cause the pain syndrome. Interoperative diagnosis, therefore, has fundamental importance; it is more important than preoperative diagnosis, because it must aim at ascertaining every possible source of irritation or compression of the root in the territory affected by the pain. It must be done still more carefully when there is no indication of a herniated disc. In this case one must aim at determining, with exactitude, other possible causes of the irritation and compression. The operation must remove every formation that can produce the syndrome.

SUMMARY

After pointing out that in a remarkable percentage of cases of lumbarsciatica the cause of radicular irritation and compression is not due to the presence of a herniated disc or not to this alone, the author enumerates all the other causes which, isolated or associated or combined with hernia of the disc, may cause the algie syndrome. He shows that the radicular irritation and compression may be caused by pathologic factors that act on the roots from the frontal, lateral and posterior directions. He analyzes the last-mentioned factors with particular emphasis on alteration of the yellow ligaments, which until now has been interpreted as hypertrophy of these ligaments in the anterior direction.

Finally, the author, while giving all due importance to preoperative diagnosis of the cause of the irritation and radicular compression, insists that the establishment by preoperative test of the existence of irritation and radicular compression is sufficient justification for surgical intervention. From this point of view he emphasizes the importance of interoperative diagnosis of the cause of irritation and radicular compression, since intervention must be decided on this basis and must aim at creating a space ample enough for the passage of the roots involved.

RIASSUNTO

Dopo aver sottolineato che in una notevole percentuale di casi di sciatica lombare la causa dell'irritazione e della compressione non è dovuta solamente alla presenza del disco erniato, l'autore enumera tutte le altre cause che sia isolate che associate o combinate alla ernia del disco possono causare tale sindrome dolorosa. Egli dimostra che l'irritazione e la compressione radicolare possono essere date da fattori patologici agenti sulle radici in direzione frontale, laterale e posteriore, ed analizza i suddetti fattori con particolar riguardo alla alterazione dei ligamenti gialli finora interpretata come ipertrofia di questi ligamenti in direzione anteriore. Infine l'autore, pur annettendo la dovuta importanza alla diagnosi preoperatoria per ciò che riguarda la causa della irritazione e compressione radicolare, insiste sul fatto che è già una sufficiente giustificazione all'intervento chirurgico l'aver stabilito, con tests preoperatori, l'esistenza di una irritazione e compressione radicolare. Partendo da tale presupposto è molto importante la diagnosi sulle cause della irritazione e compressione radicolare fatta durante l'intervento che deve, perciò, essere condotto in modo che tutte le radici interessate siano chiaramente visualizzate.

RÉSUMÉ

Après avoir souligné que dans un pourcentage élevé de cas de sciatique lombaire, la cause de l'irritation et de la compression radiculaires n'est pas due à la présence d'une hernie discale, ou du moins pas en totalité, l'auteur énumère tous les autres facteurs qui, isolés ou associés, ou combinès à la hernie discale, peuvent provoquer le syn-

drome douloureux. Il démontre que l'irritation et la compression radiculaires peuvent être provoquées par des facteurs pathologiques agissant sur les racines dans différentes directions (frontale, latérale, postérieure). Il analyse ces derniers facteurs, en insistant sur l'altération des ligaments jaunes qui jusqu'ici a été interprétéé comme une hypertrophie antérieure de ces ligaments.

Pour terminer, et après avoir accordé toute l'importance due au diagnostic préopératoire de la cause de l'irritation et de la compression radiculaires, l'auteur insiste sur le fait que la seule présence d'un tel état constaté par des examens pré-opératoires, justifie une intervention chirurgicale. Il souligne à cet égard la valeur du diagnostic inter-opératoire quant à l'origine de cette affection. C'est sur cette base que sera décidée l'intervention qui doit tendre à créer un suffisant pour le passage des racines atteintes.

ZUSAMMENFASSUNG

Nach einem Hinweis auf die Tatsache, dass in einem bemerkenswerten Prozentsatz der Fälle von Lendenischias die Ursache der Reizung und Kompression der Nervenwurzeln nicht oder nicht ausschliesslich in einem Vorfall der Zwischenwirbelscheibe zu suchen ist, zählt der Verfasser alle anderen Ursachen auf, die allein oder in Verbindung mit einem Zwischenscheibenprolaps die schmerzhaften Erscheinungen hervorrufen können. Er zeigt, dass die Reizung und Kompression der Nervenwurzeln durch Krankheitsbedingungen entstehen können, welche die Wurzeln aus verschiedenen Richtungen her, nämlich von vorn, von der Seite oder von hinten, beeinflussen. Er hebt in diesem Zusammenhang besonders die Veränderungen der Ligg. flava hervor, die bisher als Hypertrophie der Bänder in der Richtung nach vorn aufgefasst wurden. Schliesslich betont der Verfasser, dass, so wichtig die präoperative Erkennung der Ursache der Wurzelreizung und- kompression ist, die blosse Feststellung des Vorliegens einer solchen ausreicht, um einen chirurgischen Eingriff zu rechtfertigen. Unter diesen Umständen ist es natürlich von grösster Wichtigkeit, die Ursache der Wurzelreizung und-kompression während der Operation aufzudecken, weil die Art des Eingriffs, der die Schaffung von genügend Raum zur freien Passage der Wurzeln zum Ziel haben muss, von der Diagnose abhängt.

RESUMEN

Después de señalar que en alto porcentaje de casos de ciática lumbar, la causa es la presencia de un disco herniado o por lo menos no solo eso, el autor enumera todas las otras causas, que aisladas, asociadas ó combinadas con la hernia del disco pueden causar el síndrome doloroso. El dice que la irritación y compresión radicular pueden ser causadas por factores patológicos que actúan sobre las raíces en todas direcciones: anterior, posterior y lateral. El analiza los factores mencionados, con particular énfasis en las alteraciones de los ligamentos amarillos, las cuales, hasta ahora, han sido interpretadas como hipertrofia de estos ligamentos en dirección anterior. Finalmente, el autor, mientras da toda la debida importancia al diagnóstico preoperatorio de la causa de la irritación y compresión radicular, insiste en que si se comprueba la existencia de irritación y compresión radicular por medio de pruebas preoperatorias, es suficiente para justificar la intervención quirúrgica. Desde este punto de vista él enfatiza la importancia del diagnóstico interoperatorio de la causa de la irritación y compresión radicular ya que la intervención debe decidirse sobre estas bases y debe tender a crear un espacio suficientemente amplio para permitir el paso de las raíces afectadas.

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In science the man of real genius is the man who invents a new method. The notable discoveries are often made by his successors, who can apply the method with fresh vigor, unimpaired by the previous labor of perfecting it; but the mental caliber of the thought required for their work, however brilliant, is not so great as that required by the first inventor of the method. There are in science immense numbers of different methods, appropriate to different classes of problems; but over and above them all, there is something not easily definable, which may be called *the* method of science.

The kernel of the scientific outlook is a thing so simple, so obvious, so seemingly trivial that the mention of it may almost excite derision. The kernel of the scientific outlook is the refusal to regard our own desires, tastes, and interests as affording a key to the understanding of the world.

—*Russell*

Obstetric and Gynecologic Surgery

Idiopathic Thrombocytopenic Purpura in Pregnancy

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PREGNANCY is occasionally complicated by thrombocytopenic purpura. This review of the literature will be limited to cases of true idiopathic thrombocytopenic purpura. Seventy-nine pregnancies involving 58 mothers have been reported to date. To this are added 5 pregnancies involving 4 mothers observed at Baylor University Hospital (Table 1). We shall not discuss the diagnostic criteria or the treatment of idiopathic thrombocytopenic purpura, but rather the outcome as regards the mother and infant involved in this disease.

REPORTS OF CASES

On four occasions at Baylor Hospital, idiopathic thrombocytopenic purpura in the mother has been observed as a complication of pregnancy. In each instance, splenectomy has been performed during pregnancy. There have been no maternal or fetal deaths.

CASE 1.—Mrs. E. B. J., aged 30, was hospitalized at the close of the seventh month of her fourth pregnancy. There had been one earlier normal pregnancy; one spontaneous abortion, and a seven-month stillbirth, prior to the first clinical evidence of purpura at the age of 27. For two and one-half years, purpuric spots on the legs and other surfaces had been noted intermittently. During the seventh month of the present pregnancy, generalized purpura developed. There was no familial history of hemorrhagic disease.

Examination on admission to the hospital disclosed many petechiae, of general distribution. The uterus was enlarged to a seven-month gestation. The fetal heart tones were audible, and fetal movement was present. There was no evidence of abnormal bleeding.

On admission the erythrocyte count was 3,700,000 per cubic millimeters of blood, with 11.85 Gm. of hemoglobin. The platelet count was 22,000. The leukocytes numbered 10,750 per cubic millimeter. The differential count was not remarkable. Studies of the bone marrow revealed adequate numbers of megakaryocytes; the clotting time was seventeen minutes and twenty seconds (Lee-White); the bleeding time, four minutes and forty-five seconds; the prothrombin, 100 per cent of normal; factor V, 100 per cent of normal; and the heparin titration, greatly increased.

Splenectomy was performed on the day after admission. A spleen weighing 110 Gm. was removed. Freshly drawn blood totaling 1,000 cc. was given during the operation. Moderate oozing was encountered during the early stages of the procedure; this had undergone complete cessation at the time of closure.

On the day of operation the platelet count

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rose to 82,280, and the heparin titration declined almost to normal. Convalescence was uneventful, and the patient left the hospital one week after the operation, at which time the platelet count was 250,800.

She returned to her home in another city, where, seven weeks after splenectomy, she was delivered of a male infant weighing 8 pounds and 6 ounces (3,799 Gm.). Neither the mother nor the child showed any evidence of abnormal bleeding. No blood studies are available on the infant. Mother and child remain well five years after the operation.

CASE 2.—Mrs. B. D. R., a primigravida, was admitted after approximately four months' gestation because of the recent appearance of petechiae of generalized distribution. Symptoms of purpura (petechiae, nosebleed, menorrhagia, etc.) had been observed at intervals since the age of 7, and she had formerly been told that pregnancy was inadvisable. There was no family history of hemorrhagic disease.

Examination revealed petechiae distributed generally over the body. There was no active bleeding. The uterus was enlarged to the size of a four months' gestation.

On admission to the hospital the platelets numbered 10,980; the heparin titration was moderately increased; the clotting time was seventeen minutes (Lee-White); the bleeding time, nine minutes and twenty-nine seconds. The red blood cell count was 3,660,000 per cubic millimeter, with 11.3 Gm. of hemoglobin. The leukocyte count was 8,200, with a normal differential count. The prothrombin time was 100 per cent of normal; there was full bone marrow with normal numbers of megakaryocytes.

Splenectomy was performed five days after admission. The patient was given 1,000 cc. of fresh blood during the procedure. A spleen weighing 150 Gm. was removed. Bleeding was severe prior to ligation of the splenic vessels, after which it abated promptly. The platelets count rose to 67,750 on the day of operation and reached a level of 390,400 by the second postoperative day. Prompt recovery ensued, and the patient was discharged from the hospital on the seventh day.

She returned to her home in a rural community, where, five months after splenectomy, she was delivered, spontaneously, at term, of a boy weighing 7 pounds (3,175 Gm.). There was no abnormal bleeding. The child appeared normal at birth, but petechiae developed some four hours later. During the first week some blood was observed in the infant's stools. He recovered spontaneously, and has shown no evidence of purpura six years after birth. No blood studies are available.

Four months after delivery the mother underwent appendectomy, without evidence of abnormal bleeding. This operation also was performed elsewhere.

The patient was then divorced from her first husband and remarried. Some time thereafter, she became pregnant for the second time. She was seen here during the fifth month of pregnancy and was found to have a platelet count of 115,500. She had shown no hemorrhagic tendency at any time since the splenectomy. She returned home, where she was spontaneously delivered at term, without evidence of abnormal bleeding.

The infant, a girl, weighed 7 pounds (3,175 Gm.) and appeared normal at birth. Petechiae appeared on the day after birth, and no platelets could be detected in peripheral blood smears taken at this time. A transfusion of 50 cc. of whole blood was given, after which the petechiae faded and the platelet count rose to 150,000. During the ensuing week the platelet count dropped to 80,000, and fresh petechiae appeared. The infant was then returned here and hospitalized nineteen days after birth. Generalized petechiae were present at that time.

On admission the platelets numbered 8,980; the red blood cells, 4,490,000 per cubic millimeter, with 13.4 Gm. of hemoglobin. The white blood cell count was 8,050 per cubic millimeter, and the differential count normal. The clotting time was eight minutes; the bleeding time, four minutes. There was full bone marrow with adequate megakaryocytes.

Whole blood transfusions were given, and ACTHAR gel. was administered in doses of 10 to 20 mg. daily. The daily platelet counts remained around 10,000.

Because of failure of the child to make any satisfactory hematologic response after three weeks of the aforementioned treatment, it was decided to perform splenectomy. This was done on the forty-fourth day after birth. Mild bleeding was encountered early in the procedure but ceased after ligation of the splenic vessels. Within twenty-four hours the platelet count had risen to 56,430, and by the third day, it had reached a level of 345,100. The infant left the hospital five days after operation.

TABLE 1.—*Cases of Thrombocytopenic Purpura in Pregnancy*

			Mother						Infant	
Author and Year of Report	*Para and Gravida*	*Splenectomy? When?*	*Outcome*	*Type of Delivery*	*Platelet Count at Delivery*	*Postpartum Hemorrhage*	*Platelet Count at Birth*	*Type and Duration of Symptoms*	*Duration of Gestation*	*Outcome*
Waltner, 1924	P 1 G 2	None		Spontaneous	60,000	Yes	90,000	Purpura	Prem.	Recovered
Liebling, 1926	P 0 G 1	None	Cured in 3 mo.	Spontaneous	1 week pp 20,000		40,000	Numerous petechiae and bloody vomitus 3 weeks	Term	Recovered
Shands, 1930	P 2 G 3	Yes 6½ mo.	Good	Cesarean	95,000	No			Prem. 6½ mo.	Died 3 hours
Siegler, 1934	P 3 G 5	None	Cured	Spontaneous	80,000	Yes		None	Term	Died 4th day Cause?
DeSaussure and Townsend, 1935	P 8 G 9	None		Spontaneous	33,000	No		None	Term	Stillborn
Sanford, Leslie, and Crane, 1936	P 0 G 1	None		Spontaneous	18,000	No	20,000	Purpuric rash for 10 days	Term	Recovered
Troland and Lee, 1936	P 0 G 1	3 mo. pp	Cured	Spontaneous	78,000	No		None	Term	Normal
Davidson, 1937	P 0 G 1	10 yr. before	Recurred	Spontaneous	21,000	Yes	56,000	Petechiae ecchymoses 2½ mo.	Term	Recovered
Bernstein, Newman and Hitzig, 1939	P 0 G 1	5 mo. gestation	Cured	Spontaneous	Normal	No	170,000	None	7 mo. 1,361 Gm.	Neonatal death—Premature
Israel and Winson, 1941	P 0 G 1	None	Good	Spontaneous		Yes			6 mo. triplets	Two stillborn, macerated; 3d died 3 hr.
Urbanski and Hunter, 1942	P 0 G 1	1 mo. pp	Symptomatic cure of chronic T.P.	Spontaneous	50,000	No		General petechiae and hemorrhage	Term	Stillborn
	P 1 G 2	2 yr. before		Spontaneous	60,800	No	23,000	Mild petechiae 3 days	Term	Recovered

TABLE 1.—*Cases of Thrombocytopenic Purpura in Pregnancy*—continued

				Mother					Infant	
Author and Year of Report	*Para and Gravida*	*Splenectomy? When?*	*Outcome*	*Type of Delivery*	*Platelet Count at Delivery*	*Postpartum Hemorrhage*	*Platelet Count at Birth*	*Type and Duration of Symptoms*	*Duration of Gestation*	*Outcome*
Whitney and Barritt, 1942	P 0 G 1	12 yr. before	Recurred	Low forceps	70,000	No	80,000	Many purpural spots	Term	Died 3d day, intracranial hemorrhage
Burnett and Klass, 1943	P 0 G 1	None		Spontaneous	76,000	No	332,000	None	Term	Normal
Phythyon and Lartz, 1943	P 0 G 1	8 mo. gestation	Cured	Low forceps	168,000	No			8 mo.	Stillborn; macerated
Finn, 1944	P 2 G 3	3 yr. before	Cured	Spontaneous		No		None	Term	Normal
Finn, 1944	P 0 G 1	9 yr. before	Recurred at pregnancy	Spontaneous	45,000	No	120,000	Many petechiae; cephalo-hematoma	Term	Died 3d day; subarachnoid hemorrhage
Finn, 1944	P 0 G 1	3 yr. before	Recurred at pregnancy	Spontaneous		No		None	Term	Normal
Morrison and Samwick, 1945	P 0 G 1	None		Spontaneous	40,000	No	40,000	Purpura and rectal bleeding 3 wks.	Term	Recovered
Dinsmore and Dutlinger, 1946	P 0 G 1	8 mo. gestation	Cured	Cesarean	70,000	No		None	8 mo.	Stillborn; macerated (premature)
Patterson, 1946	P 0 G 1	None		Low forceps	60,000	No	18,750	Blood in stools; ecchymoses 75 days	Term	Recovered
Wolff and Limarzi, 1946	P 0 G 1	1 mo. pp	Cured	Spontaneous	Normal	No		None	Term	Normal
Talmadge and Berman, 1947	P 0 G 1	3 yr. before	Recurred at pregnancy	Spontaneous	25,000	No	9,000	Mild for 5 wks.	Term	Recovered
	P 1 G 2	7 yr. before		Spontaneous	64,000	No	5,000	Mild for 2 mo.	Term	Recovered
	P 2 G 3	8 yr. before		Spontaneous	9,000	No		Mild for 2 mo.	38 wks.	Recovered

TABLE 1.—*Cases of Thrombocytopenic Purpura in Pregnancy*—continued

Author and Year of Report	Para and Gravida	Splenectomy? When?	Outcome	Mother: Type of Delivery	Mother: Platelet Count at Delivery	Mother: Postpartum Hemorrhage	Mother: Platelet Count at Birth	Mother: Type and Duration of Symptoms	Infant: Duration of Gestation	Infant: Outcome
Barnes and Doan, 1948	P 0 G 1	5 mo. gestation	Cured	Low forceps	Normal	No	Normal	None	Term	Normal
Barnes and Doan, 1948	P 0 G 1	7 mo. gestation	Cured	Spontaneous	Normal	No		None	Term	Normal
Chalmers, 1948	P 1 G 2	None	Cured	Spontaneous	Normal	No	346,000	None	Term	Normal
Arrowsmith, 1949	P 0 G 3	At section	Cured	Cesarean		No	100,000	None	Term	Normal
Stroebel, Campbell and Hagedorn, 1949	P 0 G 3	36 wk. gestation	Symptoms cured; chronic T.P.	Low forceps	213,000	No	88,000	T.P. for 2 wks.	Term	Recovered
Stroebel et al., 1949	P 0 G 1	6 yr. pp	Recurred			No	Decreased	Purpura	Term	Recovered
Epstein, 1950	P 0 G 1	None		Spontaneous	100,000	No	188,000	Few petechiae	Term	Recovered
Epstein, 1950	P 2 G 3	None		Spontaneous	70,000	None	63,000	Mild T.P. 2 mo.	Term	Recovered
Epstein, 1950	P 0 G 1	7½ mo. gestation	Cured	Low forceps	200,000	No	13,000	For 2 yr.	Term	Recovered
Epstein, 1950	P 0 G 1	2 yr. before	Cured	Spontaneous		No	Decreased	None	Term	Recovered
Epstein et al., 1950	P 0 G 1	5 yr. before	No symptoms	Cesarean	20,000	No	8,000	Melena from 6 wk. to 2 mo.	Term	Recovered
	P 1 G 2	6 yr. before	Chronic T.P.	Cesarean	20,000	No	11,000	Petechiae for 3 mo.	Term	Recovered
	P 2 G 3	11 yr. before	Chronic T.P.	Cesarean	10,000	No	13,000	Petechiae for 3 mo.	Term	Recovered
Morris, 1950	P 1 G 2	None	Chronic T.P.	Spontaneous		Yes	35,000	Few petechiae for 9 wks.	Term	Recovered

TABLE 1.—*Cases of Thrombocytopenic Purpura in Pregnancy*—continued

Author and Year of Report	Para and Gravida	Splenectomy? When?	Mother: Outcome	Mother: Type of Delivery	Mother: Platelet Count at Delivery	Mother: Postpartum Hemorrhage	Mother: Platelet Count at Birth	Mother: Type and Duration of Symptoms	Infant: Duration of Gestation	Infant: Outcome
Robson and Davidson, 1950	P 0 G 1	7 mo. T.P.	Recurred	Spontaneous		No	210,000	None	Term	Normal
Robson and Davidson, 1950	P 0 G 1	6 wk. pp	Symptoms cured	Spontaneous	25,000	No	130,000	Purpura for 3 days	Term	Recovered
Shipton, 1950	P 0 G 1	2½ mo. gestation	Recurred		120,000	No		None	7 mo. (premature)	Recovered
Barnes, Wiseman and Zollinger, 1950	P 0 G 1	7 mo. gestation	Cured	Low forceps	Normal	No		None	Term	Normal
Glick and Weiser, 1951	P 1 G 2	6 mo. gestation	Died 12 days pp	Spontaneous	50,000	No			6 mo.	Stillborn; macerated
Mallia and Carpentier, 1951	P 0 G 1	None		Low forceps		No		None	Term	Recovered
	P 1 G 2	16 day pp	Cured	Spontaneous	37,000	No		None; twins	Term	Normal
Mallia and Carpentier, 1951	P 0 G 1	None		Low forceps	130,000	No	Normal	None	38 wks.	Normal
	P 1 G 2	None		Spontaneous	110,000	No	Normal	None	Term	Normal
Slaughter, Southwich, Falls and Limarzi, 1951	P 1 G 3	At delivery	Symptoms cured; chronic T.P.	Cesarean	2,600	No	50,000	None	Term	Normal
Hammond, 1952	P 0 G 1	None		Spontaneous		No		None	Term	Normal
	P 1 G 2	None		Spontaneous	90,000	No		None	Term	Normal
Univ. of Minnesota, 1952	P 0 G 1	8 yr. before	Recurred	Spontaneous		No			Term	
	P 1 G 2	9 yr. before		Spontaneous	36,000	No		Petechiae	Term	Recovered

TABLE 1.—*Cases of Thrombocytopenic Purpura in Pregnancy*—continued

Author and Year of Report	Mother: Para and Gravida	Mother: Splenectomy? When?	Mother: Outcome	Mother: Type of Delivery	Mother: Platelet Count at Delivery	Mother: Postpartum Hemorrhage	Infant: Platelet Count at Birth	Infant: Type and Duration of Symptoms	Infant: Duration of Gestation	Infant: Outcome
	P 2 G 3	11 yr. before		Breech	38,000	No	54,000	T.P. 2½ mo.	Term	
	P 3 G 4	12 yr. before		Spontaneous	32,000	No	52,000		Term	Normal
Harrington et al., 1953	P 1	2 mo. pp	Recurred	Spontaneous	165,000	No	None	Hemorrhage, petechiae 12 wks.	Term	Recovered
	P 2	1 yr. before			620,000	No	20,000	None, T.P. 11 wks.		Recovered
Harrington et al., 1953	P 1	6 mo. gestation	Cured		1,235,000		18,000	Petechiae 4 days		Recovered
Harrington et al., 1953	P 0 G 1	2 yr. before		Spontaneous	8,000	No	0	Petechiae, ecchymoses 11 wks.	Term	Recovered
	P 1			Spontaneous	36,000	No	0	blood in stools, 7 wks.	Term	Recovered
	P 2			Spontaneous	7,000	No	0	Petechiae 9 wks.	Term	Recovered
	P 3			Spontaneous	0	Yes	0	Petechiae, ecchymoses 7 wks.	Term	Recovered
	P 4			Spontaneous	0	No	17,000	Petechiae, ecchymoses 9 wks.	Term	Recovered
Harrington et al., 1953	P 3	7 mo. gestation	Recurred				268,000	None		Normal
Harrington et al., 1953	P 2	9 mo. gestation	Recurred	Cesarean	30,000	Yes	270,000	None		Normal
Peterson and Larson, 1954	P 2 G 4	3 mo. before pregnancy	Recurred	Cesarean	450,000	No		None	Term	Normal
Chalmers, 1954	P 0 G 1	None	Mild chronic T.P.	Low forceps		No	45,000	4 days	Term	Recovered
Chalmers, 1954	P 0 G 1	None	Cured	Spontaneous	114,000	No	33,000	10 days	Term	Recovered

TABLE 1.—*Cases of Thrombocytopenic Purpura in Pregnancy*—continued

Author and Year of Report	Mother: Para and Gravida	Mother: Splenectomy? When?	Mother: Outcome	Mother: Type of Delivery	Mother: Platelet Count at Delivery	Mother: Postpartum Hemorrhage	Mother: Platelet Count at Birth	Mother: Type and Duration of Symptoms	Infant: Duration of Gestation	Infant: Outcome
Newmark, 1955	P 1 G 3	5 mo. gestation	Remission	Spontaneous		No		None	37 wks.	Normal
Smith, 1955	P 0 G 1	14 mo. pp	Good	Spontaneous		Yes			7 mo.	Stillborn
	P 1 G 2	1½ yr.	Good	Cesarean	225,000	No			8 mo.	Stillborn; abruptio placentae
	P 2 G 3	3 yr.	Good	Spontaneous	167,000	No		None	Term	Normal
Coopersmith, B. I., 1955	P 1 G 2	6 mo. gestation	Good			No		None	Term	Normal
Coopersmith, B. I., 1955	P 0 G 1	3 mo. gestation	Good			No		None	Term	Normal
Schoen, 1956		2½ yr. before	Mild chronic T.P.	Low forceps	90,000	No	14,000	Generalized petechiae 3 mo.	Term	Recovered
Kaye and Dufault, 1957	P 1 G 3	None	Good	Spontaneous	309,000	No	110,600	None	Term	Normal
Mendel and Sparkman, 1957	P 1 G 4	7 mo. gestation	Cured 5 yr.	Spontaneous		No		None	Term	Normal
Mendel and Sparkman, 1957	P 0 G 1	4 mo. gestation	Cured 6 yr.	Spontaneous		No		Petechiae, blood in stools 7 days	Term	Recovered
Mendel and Sparkman, 1957	P 1 G 2	2 yr.	Cured	Spontaneous		No	None	General petechiae severe splenectomy 44th day	Term	Recovered
Mendel and Sparkman, 1957	P 2 G 3	4½ gestation	Recovered	Spontaneous	200,000	No	500,000	None	Term	Normal
Mendel and Sparkman, 1957	P 0 G 1	5 mo. gestation	Recovered	Low forceps	600,000	No	400,000	Few petechiae at 4 hr.	Term	Recovered

Mother and child remain well four years after delivery.

CASE 3.—Mrs. L. M., aged 30, first showed evidence of purpura two weeks prior to admission after four and one-half months of her third pregnancy. Each of the two previous pregnancies had terminated in normal delivery of healthy infants. The past history and the family history revealed no hemorrhagic disease. At the time of admission the patient was bleeding from the nose and gums and exhibited generalized purpura. The uterus was enlarged to a size consistent with five months' gestation, and the fetal heart tones were audible.

On admission, the platelets numbered 4,500; and the red blood cells 900,000 per cubic millimeter, with 3.75 Gm. of hemoglobin. The leukocyte count was 20,000, with 3 per cent normoblasts, 4 per cent myelocytes, 3 per cent young forms, 26 per cent band forms, 51 per cent segmented forms, 11 per cent lymphocytes, and 5 per cent monocytes. The bleeding time was ten minutes and thirty-one seconds; the clotting time, in excess of fifteen minutes; the bone marrow was hyperplastic and contained megakaryocytes in normal amounts; heparin titration showed pronounced increase.

The initial treatment consisted of multiple fresh blood transfusions, 1,000 cc. daily, with 15 to 20 mg. of ACTH in each pint of blood. There was no response. Splenectomy was performed on the third day after admission. The spleen weighed 310 Gm. Although the operation was completed without severe loss of blood, there was no significant platelet response, and the patient remained under treatment in the hospital for six weeks after the operation. Throughout this long period she was given almost daily transfusions of 500 to 1,000 cc. of whole blood or an equivalent amount of packed cells and platelet concentrate. ACTH was continued, plus antibiotics and Vitamin C, throughout this period. The platelet counts rarely exceeded 20,000 and were usually much lower. At intervals gross hematuria and bloody stools were observed, in addition to continual bleeding from the nose and gums. Protamine sulfate was given on two occasions during severe episodes of bleeding. Gradual cessation of bleeding finally occurred, and the patient was discharged fifty-nine days after admission. At the time of discharge, the platelet count was only 9,870.

Four months after the splenectomy, the patient was admitted to the obstetric service at term. She had remained free of hemorrhagic symptoms in the interval. Laboratory studies disclosed 192,700 platelets; 4,000,000 red blood cells per cubic millimeter, with 13.9 Gm. of hemoglobin. The clotting time was eleven minutes and fifty-two seconds (Lee-White); the bleeding time, two minutes and forty seconds. Fragility was within normal range; reticulocytes, 1.4 per cent; white blood cells, 11,600 per cubic millimeter, with a normal differential count.

A living microcephalic twin was spontaneously delivered. The placenta contained a degenerated, blighted twin. No abnormal bleeding was noted in either mother or child. Immediately postpartum, the platelet count of the mother was 200,000 and that of the infant 500,000. Neither mother nor child showed any

TABLE 2.—*Mortality*

	Total	*Deaths*	*Per Cent*
Maternal	62	1	1.6
Fetal	84	16*	19

*Triplets, 1 case.

TABLE 3.—*Causes of Deaths in Infants*

Prematurity	3	
Purpura	3	
Stillbirth	9	(2 Premature triplets)
Unknown cause	1	

TABLE 4.—*Relation of Splenectomy to Mortality Rates*

		Fetal		*Maternal*	
	Total	*Deaths*	*Mortality, Per Cent*	*Deaths*	*Mortality, Per Cent*
Splenectomy before onset of pregnancies	31	4	12.9	0	0
Splenectomy during pregnancy	22	5	22.7	1	4.5
No splenectomy during or after pregnancy	31	7*	22.6	0	0

*Triplets, 1 case

TABLE 5.—*Breakdown of Infant Deaths Following Splenectomy*

Shands	1929	Cesarean section with splenectomy at 6½ months; infant died in 3 hours
Bernstein	1939	Splenectomy at 6 months; infant born 6 weeks later; birth weight 3 lbs.; died on 6th day of atelectasis and prematurity; no purpura
Phythyon	1943	Stillborn premature at 8 months by low forceps 3 days after splenectomy
Dinsmore	1946	Cesarean section 8 days after splenectomy at 8 months for abruptio placentae, with stillbirth
Glick	1951	Splenectomy at 6 months; no evidence of fetal life after operation; stillborn macerated fetus delivered on 7th postoperative day; mother died on 12th postoperative day of massive hematemesis and shock

hemorrhagic tendencies six months after delivery.

CASE 4.—Mrs. C. G., aged 34, first showed signs of purpura two days prior to admission in the fifth month of her first pregnancy. There was no history of any hematologic disorder prior to the present illness. On admission there were multiple diffuse petechiae and ecchymoses over the entire body. The lower extremities were covered with petechiae and occasional bruises. The tongue, the palate and the buccal mucosa showed petechiae, with slight oozing from the gums. The uterus was enlarged to a size consistent with five months' gestation. The fetal movements were active.

Laboratory studies on admission to the hospital revealed 14,640 platelets. The heparin titration was increased; the clotting time was two minutes and thirty seconds; the bleeding time, nine minutes and forty-seven seconds. The red blood cell count was 3,660,000 per cubic millimeter; with 11.7 Gm. of hemoglobin. The white blood cells numbered 7,600 per cubic millimeter, with a normal differential count.

She was given 500 cc. of whole blood with 15 mgm. of ACTH, and received ACTHAR gel., 40 mg., intramuscularly twice daily. On the fourth day after admission the platelet count was 65,280 and the bleeding time three minutes.

Splenectomy was performed with some difficulty, and the patient was given 4 liters of blood during the procedure. A spleen weighing 174 Gm. was removed. A few hours after the operation, the laboratory data were as follows: platelets, 105,040, hemoglobin, 12 Gm., red blood cell count, 4,040,000 per cubic millimeter. The postoperative course was uneventful, and the patient was discharged from the hospital on the fifth postoperative day, with a platelet count of 221,760.

At term a normal labor took place. The platelet count was 535,200 before delivery. By low forceps and episiotomy she was delivered of a normal boy. There was no excessive blood loss. The child's platelet count ranged from 254,250 to 501,600. At two hours, he had a few petechiae, which faded quickly. Mother and infant have shown no hemorrhagic tendencies in a three-year follow up.

Maternal and Fetal Mortality Rates.—The maternal mortality rate was low; only 1 of 62 mothers with idiopathic thrombocytopenic purpura died. In this case the mother had had mild symptoms of purpura since early childhood, with an acute exaccerbation after six months' gestation. The infant apparently died at the time of splenectomy, and on the seventh postoperative day a stillborn, macerated fetus was delivered. There was a poor postoperative platelet response, and on the twelfth postoperative day the mother died of massive hematemesis and shock (Table 2).

In this study, the maternal mortality rate was 1.6 per cent. This rate does not exceed that expected from idiopathic thrombocytopenic purpura alone. It would appear that the coexistence of pregnancy and this disease does not increase the fatal aspects of the purpura. There were 16 fetal deaths (in 1 case premature triplets were involved) with a fetal mortality rate of 19 per cent. The causes of fetal death are shown in Table 3.

Effect of Splenectomy on Pregnancies with Purpura.—Since splenectomy is a major means of therapy in cases of severe

TABLE 6.—*Relation of Splenectomy to Involvement of Fetus*

	Total	Infant Involved		Infant Normal	
		Number	Per Cent	Number	Per Cent
Mothers splenectomized before delivery	40	31	77.5	9	22.5
Mothers not splenectomized before delivery	25	16	64	9	36
Total	65	47	72.3	18	27.7

TABLE 7.—*Postpartum Bleeding*

	Total	Normal Postpartum Bleeding	Postpartum Hemorrhage
Mothers with platelet count decreased at delivery	43	38 (88.4%)	5 (11.6%)
Mothers with platelet count normal at delivery	12	12	0
Total	55	50 (90.9%)	5 (9.1%)

or prolonged idiopathic thrombocytopenic purpura, a study was made of such cases in order to determine the effect of this operation on both the mother and infant (Table 4).

There was 1 maternal postoperative death in the total group of 51 cases. One of the patients succumbed to a recurrence of purpura two years after the operation and was found to have accessory spleens.

The fetal mortality rate for pregnancies during which splenectomy was performed was 23 per cent.

A breakdown of these figures reveals that 4 of these deaths occurred in the first 4 cases reported. One was thought to be due to causes other than purpura. In the first case reported, cesarean section was performed at six and one-half months, at the same time as splenectomy, with death of a premature fetus after three hours. This can be considered an avoidable death, as no reason for section seemed indicated at that time (Table 5).

It must be emphasized that in the cases in which splenectomy was performed during pregnancy the manifestations of I.T.P. were usually more severe than in cases in which other types of therapy were employed.

It should be noted that, in most of the cases, the fetus was involved whether or

TABLE 8.—*Type of Delivery*

	No.	Per Cent	Spontaneous	Forceps
Vaginal	64	86.5	53	11
Cesarean section	10	13 5		

Résumé of Sections

1 Shands	1929	6½ months, simultaneous with splenectomy; no indication for section
2. Dinsmore et al.	1946	Premature separation of placenta with intrauterine death 8 days after splenectomy
3. Arrowsmith et al.	1949	Cephalopelvic disproportion; simultaneous with splenectomy
4. Epstein et al.	1950	Cephalopelvic disproportion, with 2 repeat sections
5. Slaughter	1951	Previous section with acute attack of purpura at term
6. Harrington	1953	Indication not given
7. Peterson et al.	1954	Premature separation of placenta
8. Smith	1955	Premature separation of placenta

not the mother had undergone splenectomy before delivery (Table 6).

Splenectomy in the mother does not always result in remission of idiopathic thrombocytopenic purpura, although it usually diminishes the purpuric manifestations of the disease. The development of neonatal thrombocytopenic purpura in the infants of mothers with this disease seems to bear no relation to the age or gravidity of the mother, or to whether or not splenectomy was performed. In those cases in which splenectomy was performed prior to the onset of pregnancy, however, the fetal mortality rate was significantly lower (13 per cent).

Postpartum Bleeding in I.T.P. — The question of postpartum hemorrhage always arises in cases of pregnancy associated with thrombocytopenic purpura. It is almost universally agreed that the bleeding from the uterus after delivery is prevented primarily by mechanical means rather than by any excessive clotting process. The contracting uterine musculature closes the uterine sinuses, thus producing hemostasis.

In the 84 pregnancies reviewed in this series, there were 7 cases of excessive postpartum bleeding, an overall incidence of 8.3 per cent. As to the cases in which data are available as to the platelet count at the time of delivery, it was noted that in no cases in which the count returned to normal at that time was postpartum hemorrhages recorded (Table 7). In the cases in which the platelet count was decreased at the time of delivery, there were 5 postpartum hemorrhages in 43 cases, an incidence of 11.6 per cent. Therefore, in all cases of idiopathic thrombocytopenic purpura, a platelet count during labor is essential, in view of the high incidence of postpartum bleeding when the count is decreased.

TABLE 9.—*Relation of Mother's Platelet Count to Involvement of Infant*

		Infant Involved		*Infant Normal*	
	Total	*Number*	*Per Cent*	*Number*	*Per Cent*
Mothers with platelet count normal at delivery	15	6	40	9	60
Mothers with platelet count decreased at delivery	41	30	73	11	27
Total	56	36	64.3	20	35.7

Type of Delivery.—The mode of delivery in this series usually followed general obstetric lines, although the section rate was higher than usual. In 75 cases the type of delivery was specified. Of these, 65 were by the vaginal route, with 54 spontaneous and 11 forceps deliveries. There were 10 cesarean sections, an incidence of 13.5 per cent (Table 8).

Involvement of the Fetus.—The symptom complex and laboratory results observed in an infant born to a mother with idiopathic thrombocytopenic purpura are similar to the mother's. Most of the infants have a mild type of thrombocytopenic purpura, which runs a self-limiting course. There are all gradations of involvement, from simple thrombocytopenia, with no purpuric manifestations, to the severest forms of the disease, with severe hemorrhagic symptoms and marked thrombocytopenia. The laboratory data are the same as for the adult. Most of the infants need only supportive therapy; an occasional case of serious involvement, however, will require vigorous therapy. In one case in the Baylor series splenectomy had to be performed on the infant on the forty-fourth day after birth. The results were excellent.

Epstein and others have theorized that congenital thrombocytopenic purpura is

the result of "transfer of an immune body, hormone, or other substance across the placental membranes which depresses platelet formation in the infant." Evans demonstrated the presence of platelet agglutinins in the serum of patients with idiopathic thrombocytopenic purpura. Schoen reported a case in which platelet agglutinins were demonstrated initially in the blood of both mother and infant but disappeared from the blood of the baby after return of the platelet level to normal. The disappearance of the agglutinins in the baby, together with their persistence in the mother, suggests that the platelet agglutinins were passively transferred from the mother to the infant across the placental membranes. This may occur whether or not operation has been performed and may explain the high percentage of infants involved in purpura at birth (Table 9). Forty per cent of the infants born to mothers with normal platelet counts at delivery and 73 per cent of those born to mothers with an abnormal count were involved.

There is one special group of cases of I.T.P. complicating pregnancy that has particular interest. In these cases the mother has shown no evidence of purpura prior to pregnancy; severe manifestations of purpura develop, coinciding with evidence suggestive of fetal death, yet all evidence of purpura disappears upon the delivery of a stillborn, macerated fetus. Although it may be argued that the fetal death in most cases was the result of hemorrhage and other disorders secondary to the purpura, the conjecture may be entertained that the purpura is a reaction to the products of conception or to death of the child in utero. If this hypothesis could be substantiated, the treatment of this select group would consist of termination of the pregnancy.

CONCLUSIONS

A review of the literature reveals 76 pregnancies involving 55 mothers with idiopathic thrombocytopenic purpura. To these the authors add 5 pregnancies in 4 mothers whose cases have not been reported heretofore.

The following observations were made:

The fetal mortality rate was 19 per cent and the maternal death rate 1.6 per cent.

An analysis of the effect of splenectomy on these pregnancies showed that the incidence of fetal death (23 per cent) was approximately the same in pregnancies in which splenectomy was performed during gestation and in those in which more conservative measures were employed. In cases in which splenectomy had preceded pregnancy the fetal death rate was decidedly lower (13 per cent).

Postpartum hemorrhage had an overall incidence of 8.3 per cent. There were no cases of excessive bleeding, however, when the platelet count had returned to normal at delivery. In cases of a decreased platelet count at delivery the incidence rose to 11.6 per cent.

The type of delivery usually followed the obstetrical indications. The rate for cesarean section was higher than normal (13.5 per cent).

Congenital thrombocytopenic purpura occurs in 40 per cent of the infants of mothers with normal platelet counts and in 73 per cent of those whose mothers show a decrease in platelet counts at delivery. The passive passage of platelet agglutinins across the placental membranes from mother to infant is discussed.

CONCLUSIONS

La littérature compte 76 cas de grossesse chez 55 femmes atteintes de purpura hémorragique. Les auteurs ajoutent 5 cas personnels (4 femmes).

Lls ont fait les constatations suivantes:

La mortalité foetale a été de 19%, celle de la mère de 1,6%.

L'étude des résultats de la splénectomie sur ces grossesses montre une fréquence d'environ 23% quand la splénectomie a été pratiqée durant la gestation, ainsi que dans ceux où des mesures plus conservatrices avaient été appliquées. Dans les cas où la splénectomie a précédé la grossesse, on note un taux de mortalité beaucoup plus faible (13%).

Le taux de l'hémorragie post-partale a été de 8,3%. Ll n'y a cependant eu aucun cas d'hémorragie excessive une fois les thrombocytes revenus à la normale au moment de l'accouchement. Lors de diminution des thrombocytes au moment de l'accouchement, la fréquence est montée à 11,6%.

Les accouchements ont en général pu être pratiqués normalement, mais le taux d'indication de la césarienne a été plus élevé (13,5%).

le purpura hémorragique congénital se rencontre chez 40% des nouveaux-nés de mères ayant un taux normal de thrombocytes, et chez 73% de ceux dont la mère présente à l'accouchement un taux de thrombocytes abaissé. Le passage passif Vde la mére á l'enfant d'agglutinines provenant des thrombocytes, à travers les membranes du placenta, est discuté.

CONCLUSIONI

In una rassegna della letteratura sono stati trovati 76 casi di gravidanza in donne con porpora trombopenica idiopatica. Ne vengono aggiunti, dagli autori, altri 5 casi originali.

Si fecero le seguenti considerazioni:

La mortalità fetale fu del 19 per cento e quella materna del 1,6.

Un'indagine sugli effetti della splenectomia dimostrò che la mortalità fu di circa il 23% nelle gravide in cui la splenectomia era stata fatta durante la gestazione o non era stata fatta, mentre fu del 13% soltanto in quelle che erano state splenectomizzate prima della gravidanza.

L'emorragia post, partum si verificò più spesso (8,3%), in forme tuttavia non gravi, in quei casi in cui la conta delle piastrine era tornata a dare valori normali al momento del parto; nei casi, invece, in cui persisteva piastrinopenia, l'aumento della frequenza dell'emorragia salì all'-11,6%.

Il tipo del parto usualmente avvenne secondo le indicazioni ostetriche; il numero dei parti per via cesarea fu più alto della norma (13,5%).

La porpora trombopenica congenita è riscontrabile nel 40% dei nati da madri con numero di piastrine normali, e nel 73% dei nati da madri con piastrinopenia al momento del parto. Viene discusso il problema del passaggio delle agglutinine piastriniche dalla madre al feto attraverso il filtro placentare.

CONCLUSIONES

Revisndo la literatura se encuentrar 76 embarazos en 55 madres afectas de púrpura trombocitopénica idiopática. Los autores añaden cinco embarazos en cuatro madres que no han sido publicados hasta ahora. Se han podido hacer las siguientes observaciones: la mortalidad fetal fué de un 19% y la maternal de un 16%; en un 23% de los casos fué practicada la esplenectomía. En los casos en las cuales la esplenectomía se hizo entes de la gestación, el tanto por ciento de muertes fetales disminuyó notablemente (13%). La hemorragia post-partum se presentó en 18.3% de los casos. No hubo casos de hemorragia excesiva cuando el recuento de plaquetas había vuelto a hacerse normal en el momento del parto. En los casos de recuento de plaquetas descendido en el momento del parto, los casos de hemorragia subieron a un 11,6%.

El tipo de parto generalmente fué conforme a las indicaciones obstétricas. La cifra de cesáreas fué más elevada que normalmente 13,5%).

La púrpura trombocitopénica se presenta en un 40% de los niños de madres cuyo recuento de plaquetas es normal y en un 73% en aquellos de madres con un recuento de plaquetas bajo en el momento del parto. Se discute por último el paso de las aglutinas de la plaquetas de la madre al hijo a través de las membranas placentarias.

SCHLUSSFOLGERUNGEN

Eine Durchsicht der Literatur ergibt 76 Schwangerschaften bei 55 Müttern mit idiopathischer thrombozytopenischer Purpura. Diesen fügen die Verfasser fünf bisher unveröffentlichte Fälle von Schwangerschaft bei vier Müttern hinzu.

Es kam zu folgenden Beobachtungen:

Die Sterblichkeitsquote der Neugeborenen betrug 19 Prozent, die der Müttern 1,6 Prozent.

Eine Untersuchung des Einflusses der Milzresektion bei diesen Schwangerschaften ergab, dass die Sterblichkeit der Neugeborenen etwa 23 Prozent betrug, wenn die Milzresektion während der Schwangerschaft ausgeführt wurde und wenn konservativere Massnahmen angewandt wurden. Wo die Milzresektion vor Eintritt der Schwangerschaft vorgenommen wurde, war die Sterblichkeit der Neugoborenen entschieden geringer (13 Prozent).

Blutungen nach der Entbindung traten in 8,3 Prozent aller Fälle auf. Uebermässige Blutungen wurden jedoch niemals beobachtet, wenn die Zahl der Blutplättchen zur Zeit der Entbindung einen normalen Wert erreicht hatte. War die Zahl der Blutplättchen bei der Entbindung herabgesetzt, so stieg das Vorkommen von Blutungen nach der Entbindung auf 11,6 Prozent.

Ueber die Art der Entbindung wurde gewöhnlich auf Grund der allgemeinen geburtschilflichen Indikationen entschieden. Die Zahl der Kaiserschnitte war höher als unter normalen Verhältnissen (13,5 Prozent).

Die angeborene thrombozytopenische Purpura kommt in 40 Prozent bei Kindern vor, deren Mütter eine normale Anzahl von Blutplättchen aufweisen; bei 73 Prozent der Kinder zeigen die Mütter eine Herabsetzung der Blutplättchenzahl zur Zeit der Entbindung. Die Verfasser erörten die passive Passage von Blutplättchenagglutininen durch die Plazentarmembran von der Mutter zum Kind.

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Maternal Mortality and Morbidity from Anesthesia

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IMPROVEMENT in maternal care during the past few decades has been consistent and outstanding. The steady decline in maternal mortality marks one of the most important milestones in medical progress. Credit for this is due to obstetricians, general practitioners, public health workers and many others.

The most common causes of maternal mortality are hemorrhage, infection, toxemia and heart disease. Fifth as a cause of maternal mortality is anesthesia. The first four are coming under control and, therefore, are responsible for fewer maternal deaths than formerly. Hemorrhage is less of a problem because of the greater availability and safety of blood transfusions. Infection is reduced by cleanliness and antibiotics. Deaths due to toxemia and heart disease are kept in check by better overall prenatal care.

The reduction in the major causes of maternal mortality serves to bring anesthesia into sharper focus as a cause of maternal death. According to the National Office of Vital Statistics, there are over 4,000,000 births in the United States each year, and the average maternal mortality rate for the ten-year period from 1944 to 1953 was 12.2 per 10,000 births, or about 5,000 maternal deaths per year. If 3 to 10 per cent of these deaths are due to anesthesia, as most of the surveys would indicate, there would be 150 to 500 maternal deaths due to anesthesia in an average year.

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Although not so impressive in absolute numbers, anesthetic fatalities are mounting in proportion as deaths due to other causes decline. With the increasing prominence of maternal anesthetic deaths, public and medical opinion demand immediate correction. These deaths cannot be ignored when one realizes that nearly all are preventable and that the victim is often a healthy young woman. It must be remembered that the anesthetic death rate does not tell the whole story. It does not tell how many deaths were attributed to other causes, such as heart disease, hemorrhage and toxemia. It does not tell how many accidents that were not fatal could have been prevented if good anesthetic management had been instituted. Anesthetic deaths are often kept such a secret that even those in immediate attendance do not know that the anesthesia caused them. There is no compulsory reporting of anesthetic deaths, so the real incidence is unknown.

What are the causes of these deaths? Are obstetricians the victims of their own complacency? Since delivery after delivery is uncomplicated, are they prepared to meet such complications as may occur? Do all of Dr. X's patients receive routine medication plus a general anesthetic regardless of parity, fullness of the stomach or disease? Does Dr. X choose the anesthetic that keeps the mother from delivering until he arrives? Do obstetricians in general choose the anesthetic that takes the least time and requires the least explanation to the patient? Do they use

common sense in choosing the anesthetic for each patient?

The reports of anesthetic deaths that appear in both the medical literature and the lay press stress the mortality rate according to the anesthetic agent employed. These reports mean little, if anything, since they do not reveal the predisposing factors. Too much emphasis has been placed on the anesthetic agent; too little consideration is given to the technic of administration; to the skill, professional background, and integrity of the anesthetist, and to the attentiveness and skill of the obstetrical team, which should excel in early recognition of acute emergencies and manage them with dispatch.

Critical Evaluation of Anesthetic Deaths.—The physiologic disturbances ascribed to or associated with anesthesia may affect any organ or system. No anesthetic technic is free of morbidity any more than it is free of mortality. In a review of maternal anesthetic deaths, the Medical Society of North Carolina attributed 69 per cent of the deaths to aspiration of gastric contents, spinal shock and cardiac arrest.[1] Merrill and Hingson estimated that 100 maternal deaths per year are due to aspiration.[2]

A better understanding of the catastrophe of an anesthetic death may be achieved by evaluating and classifying what happened, why it happened and how it happened. A critical study of such deaths will indicate that certain physiologic phenomena are involved. Disruption of a vital physiologic mechanism leads to death. Certain types of physiologic disturbances tend to occur more with one kind of anesthesia than with another. We shall endeavor to classify the causes of maternal anesthetic deaths according to the physiologic mechanism involved and according to the hazards associated with each type of anesthesia.

Cardiac Arrest.—Many anesthetic deaths are attributed to cardiac arrest. This term has done much to obscure the issue and confuse the investigator. It intimates that the cause is supernatural. The term "cardiac arrest" is not a diagnosis. It is merely a statement that death has occurred; it explains nothing. In any instance of cessation of effective heartbeats there are precipitating factors. A heart is made to stop by physiologic insult. Years ago the term "status lymphaticus" was used in a similar way to describe sudden deaths without obvious cause. The term "cardiac arrest" must be relegated to the obsolete position which "status lymphaticus" occupies today.

In every instance in which "cardiac arrest" occurs, there must be a pathologic lesion or a physiologic derangement of such magnitude as to prevent the continued and effective functioning of the heart. The events which lead up to "cardiac arrest" must be critically examined to determine predisposing causes, and a postmortem examination of the tissues must be conducted for anatomic evidence of disease. In rare instances evidence may be found of unsuspected serious disease.

What are the causes of cardiac standstill or fibrillation during anesthesia? Hypoxia and acidosis of the heart are the basic causes, and they may be due to anemia, hypotension, interference with the oxygen supply or carbon dioxide removal, or impairment of the coronary circulation. The use of drugs that increase cardiac irritability, such as epinephrine, pituitrin, pitressin, chloroform, trilene and, to a lesser extent, cyclopropane, all tend to set the stage for a cardiac catastrophe. In addition, the oversedated patient is hypoxic; the inadequately sedated patient is apprehensive. Consideration must be given to the preparation of patients for anesthesia just as it is to blood chemistry, the hemoglobin level and the fluid balance. All too often, the anes-

thetic phase of the patient's preparation is overlooked.

If a cardiac catastrophe does occur there must be immediate recognition of the emergency, removal of the precipitating cause and expansion of the lungs with 100 per cent oxygen. If there is no response, cardiac massage or manual artificial circulation must be instituted without delay. The need for a preconceived plan of action is obvious.

Physiologic Disturbances Leading to Anesthetic Deaths.—The maintenance of life is dependent upon a constant supply of oxygen to the vital organs. A brief period of oxygen deprivation, measured in minutes, may be sufficient to cause fatal damage to the tissues. The oxygen supply of the body is dependent upon two physiologic systems, the respiratory and the circulatory. Almost every anesthetic death can be attributed, in the final analysis, to a disruption of one or the other or both of these vital systems.

Respiratory System.—In any discussion of respiration the alveolar ventilation is of major concern, for it is in the alveoli that actual gaseous exchange takes place. The alveolar ventilation represents the "fresh air" that reaches the lungs, and is equal to the tidal volume minus the physiologic and mechanical dead space. Any decrease in tidal volume or increase in dead space diminishes the critical factor of alveolar ventilation. This in turn causes hypoxia, with acidosis due to the diminished excretion of carbon dioxide. Reduced alveolar ventilation by one mechanism or another is responsible for a major proportion of all anesthetic deaths.

Even with adequate alveolar ventilation a respiratory death may occur. This may result from a lowering of the oxygen concentration in the atmosphere inhaled by the patient. For this reason an excess of oxygen should always be the rule, in order to provide a margin of safety to both the mother and the infant. No anesthetic should be given with less than 25 per cent oxygen. Whenever an anesthetic gas or vapor is mixed with air, it must necessarily displace some of the oxygen of the air. Low oxygen concentrations during anesthesia must be avoided at all costs.

Many factors may be responsible for a reduction in effective alveolar ventilation. There may be preexisting disease of the respiratory system, such as pneumonia, tuberculosis, pneumothorax, bronchiectasis or even pulmonary edema due to cardiac disease, all of which interfere with the alveolar ventilation and thus with oxygenation and the excretion of carbon dioxide.

The administration of a general anesthetic adds three major possibilities for respiratory difficulty: obstruction, depression and paralysis. Obstruction of the airway is the most common and the most serious of all the hazards associated with the state of unconsciousness. The obstruction may be due to local disease of the nose or throat or to a valvelike action of the tongue and epiglottis. Inflammation, tumors and foreign bodies, such as chewing gum and loose dentures, may block the air passages. Particularly during induction and in very light anesthesia does obstruction of the air passages pose a great problem to the anesthetist, for the patient may not be sufficiently anesthetized to tolerate an artificial airway. By altering the position of the patient's head or by upward traction on the mandible it is usually possible to obviate this difficulty, but not without paying the penalty of some hypoxia.

Laryngospasm as a cause of respiratory obstruction is mentioned often in the medical literature and is greatly feared. Frequently, the diagnosis is mistakenly made. We have observed many instances in which the surgeon or the obstetrician

made a diagnosis of laryngospasm, and in which some other form of respiratory difficulty actually existed. The skilled anesthetist rarely encounters serious laryngospasm. If he does, he has simple and effective means of combating this condition.

Vomiting and passive regurgitation, resulting in respiratory obstruction, are by far the most important among the causes of anesthetic deaths. The obstetric patient is particularly liable to this difficulty. In many instances she has not been properly prepared by the withholding of foods and fluids. Because a very light stage of anesthesia is desirable in obstetrics, she is frequently kept at the level in which the vomiting center of the brain is stimulated. In addition, manual pressure on the fundus of the uterus often results in regurgitation of the contents of the stomach. Fear, pain, parturition and narcotics delay the stomach's emptying. If food has been ingested after the onset of true labor, the stomach must be considered full, regardless of whether or not the patient has vomited.

An empty stomach is the only certain means of preventing regurgitation; prevention is more effective than treatment. If the obstetrician is convinced that general anesthesia will give him the best results, he must repeatedly instruct his patients to avoid the intake of food after labor begins, and he must impress upon them the dangers of violating this rule. He should have an alternate type of anesthesia for those who have eaten since the onset of labor. Most patients will realize that not only is their own safety but that of the unborn child is at stake. The doctor and the delivery room personnel must bear this constantly in mind. They must not callously place the patient in jeopardy, no matter how infrequent it has been in their experience.

The regurgitation of gastric contents may result in either mechanical obstruction due to the foreign material in the air passages, or in laryngospasm or bronchospasm. These syndromes may occur either individually or concomitantly. Bronchial irritation due to the acid foreign material produces an asthma-like condition that may vary in severity from wheezing to complete absence of respiratory exchange.

Depression of the respiratory center of the brain may be produced by any narcotic, sedative or anesthetic drug. The effects of these drugs are additive. The narcotics produce slowing of the rate of respiration, while the barbiturates and the general anesthetics produce a decrease in its depth. Superimposed upon the depression caused by drugs may be that caused by debility or exhaustion.

Paralysis of respiration may result from either regional or general anesthesia. Spinal, caudal and epidural anesthetics produce increasing paralysis of the respiratory muscles as the level of anesthesia rises. The curare-like muscle relaxants used in general anesthesia cause the muscles to become unresponsive to the respiratory drive.

Convulsions caused by eclampsia, epilepsy or reactions to local anesthetic drugs also produce interference with the ability to breathe. The muscles of respiration become tensely contracted and unable to function rhythmically. The muscles that control the air passages also contract and, by holding the soft tissues in abnormal positions, cause respiratory obstruction. Both the obstruction of the air passages and the tonic contraction of the respiratory muscles greatly interfere with the ability of the patient to breathe and with the ability of the physician to provide effective artificial respiration.

The treatment of respiratory complications during anesthesia includes removal of foreign materials from the respiratory

tract, establishment of a clear airway and maintenance of adequate ventilation by artificial respiration. If simple measures do not suffice to accomplish these aims, endotracheal intubation, bronchoscopy or tracheotomy may be needed. Calling a consultant for diagnosis or treatment may be merely a soul-satisfying formality, since a consultant is rarely immediately available. Respiratory difficulty must be corrected within three or four minutes by the person in attendance. Tracheotomy should not be delayed because of the reluctance to leave the patient with a small scar on her neck or because of lack of familiarity with a minor operative procedure. Every obstetrician and anesthetist should learn the simple details of this life-saving operation. The anesthetist, also, must be an attentive diagnostician and a competent therapist, with perfectly functioning equipment immediately available.

In dealing with respiratory difficulties, drugs are of no value. Patients in severe respiratory distress do not respond to stimulating drugs. The treatment of respiratory difficulty is mechanical. Establish a clear airway, and maintain artificial respiration by mechanical means. With adequate oxygenation, the functional abilities of both respiratory center and respiratory muscles usually return within a relatively brief period. Even if for no other reason, the use of stimulant drugs is harmful because of the delay in obtaining them and in waiting for them to act.

Circulatory System.—The circulatory system may be embarrassed by preexisting disease or by the effects of anemia and hemorrhage. Assuming that the pregnant patient with heart disease has been competently treated, serious difficulty may still develop at the time of delivery. Increased blood volume plus the demand for increased cardiac work occasioned by excitement, stress and the physical activity of labor may precipitate heart failure. In cardiac patients it is best to avoid the stress of hard labor with some form of conduction anesthesia that will eliminate the bearing-down sensation and the resultant straining. In addition, with conduction anesthesia a physiologic phlebotomy can be performed by pooling blood in the lower extremities.

To prevent the compounding of circulatory insults, one must endeavor to keep all factors within the range of normal. Anemia and blood loss should be corrected. Determination of the value for hemoglobin, the red blood cell count and the hematocrit reading give a false sense of security when done immediately after a major hemorrhage and before hemodilution occurs. Hypotension must not be tolerated in obstetrics, because of its deleterious effect upon the oxygen supply of both the infant and the mother.

Depression of the circulatory system may be caused by anoxia. It may also be caused by deep anesthesia or by the rapid administration of anesthetic agents, resulting in a form of speed shock. Specific anesthetic drugs may also cause depression or abnormality of the heart itself and predispose to circulatory catastrophes.

Paralysis of the thoracolumbar sympathetic outflow may be caused by spinal, caudal and epidural anesthesia, the extent of the paralysis depending upon the height of the anesthetic level. Removal of the sympathetic innervation of the blood vessels results in their dilatation and the pooling of blood, with hypotension as the end product. Paralysis of the sympathetic outflow creates a sudden discrepancy between the capacity of the vascular bed and the quantity of fluid within it. This is the physiologic basis of spinal shock, which has often been reported as a cause of death, with the inference that it was caused by an idiosyncrasy of the drug or the patient.

With the patient in the supine position

the gravid uterus may compress the vena cava, interfering with the return flow of blood and resulting in low cardiac output and hypotension. The decrease in size of the uterine mass and the sudden release of tension within the abdomen after delivery may permit venous pooling.

The obstetrician must be aware of the fact that a sudden elevation of blood pressure may occur at the time of delivery in a previously normotensive woman. This may be caused by the vasopressor drug administered with local or conduction anesthesia. The intravenous administration of ergot preparations also leads to hypertension. The vasopressors and the ergot derivatives potentiate each other in their effects upon the blood pressure.

We shall not attempt to discuss the treatment of cardiac disease. The treatment of circulatory emergencies usually involves the replacement of blood after hemorrhage, or the treatment of hypotension or hypertension. One should attempt to replace the blood lost with an equal amount by transfusion. If whole blood is not available blood substitutes may be used, but one should bear in mind that the blood substitutes do not have the oxygen-carrying capacity that is present in the red cells only.

The treatment of neurogenic hypotension does not include blood transfusion. This condition can usually be managed by the administration of an intravenous infusion containing a dilute solution of a vasopressor drug. The patient's blood pressure may be restored and maintained at normal levels by titration, the speed of the infusion being adjusted to the requirements of the patient as indicated by determination of the blood pressure at frequent intervals.

Elevation of the blood pressure may take place during delivery owing to inadequate pulmonary ventilation or the use of vasopressor drugs, ergot preparations and pitressin or pituitrin. It is managed by hyperventilation to remove any possible accumulation of carbon dioxide. If this is not effective, the blood pressure may be brought down by the administration of sympatholytic drugs, either by infusion or by the repeated administration of very small doses. Hexamethonium bromide, in doses of 1 mg. repeated every minute or two, produces a rapid and controllable return of the blood pressure to normal levels.

The Physiologic Hazards Associated with Particular Forms of Anesthesia.—Having discussed the physiologic aberrations that may be encountered during the course of anesthesia for a delivery, we may now briefly itemize those most likely to occur with particular forms of anesthesia.

General Anesthesia.—General anesthesia is most likely to produce difficulty involving the respiratory system. Obstruction of respiration due to the encroachment of soft tissue on the airway is the most common and may occur in every case. Active or passive regurgitation of the contents of the stomach occurs frequently and may cause immediate death, immediate nonfatal difficulty or delayed pulmonary complications.

Circulatory difficulty due to general anesthesia is relatively unusual. Speed shock due to the rapid administration of anesthetic agents may occur. This is most likely to occur with intravenous agents, because of the rapidity and ease with which they may be given. Derangements of cardiac rhythm of serious importance are relatively rare and are usually associated with anoxia, although specific drugs have a tendency to predispose the patient to this difficulty.

Of particular concern in general anesthesia for delivery is the use of sodium pentothal. Although the use of pentothal has often been questioned because of its effect upon the infant, we have observed that maternal respiratory difficulty is a far more frequent complication. Laryngo-

spasm occurs more often with this agent than with any other. Regurgitation of food produces direct stimulation of the larynx, and forceps traction produces remote stimulation. In either instance, reflex laryngospasm may be serious. In our experience, aspiration of vomitus into the tracheobronchial tree in amounts great enough to require bronchoscopy has occurred far more often with pentothal anesthesia than with any other general anesthetic.

Conduction Anesthesia.—Spinal, caudal and epidural anesthesia tend to create difficulties with the circulatory system more often than with the respiratory system. During attempts to induce caudal or epidural anesthesia, massive spinal anesthesia may be produced inadvertently, resulting in hypotension and respiratory paralysis. Even when the level of conduction anesthesia is limited to the tenth thoracic segment as desired, severe hypotension may occur. This should be treated by immediate elevation of the legs, to provide an autotransfusion, and by the infusion of a vasopressor drug. Frequent observations of the blood pressure must be made in order to prevent wide fluctuations in the blood pressure. If the infusion runs too rapidly, severe hypertension may replace the previous hypotension. The use of intramuscular vasopressors cannot be relied upon for the treatment of hypotension. The vasopressors are not absorbed from intramuscular sites if hypotension is present. When the vascular system returns to normal, hypertension may develop as a result of late absorption.

The respiratory complications of conduction anesthesia are usually simply managed by mechanical artificial respiration with oxygen until the effect of the drug wears off. The administration of respiratory stimulants do no good in restoring effective respiration if the respiratory muscles are paralyzed; the stimulant drugs act upon the medulla, which is functioning unimpaired. It must be kept in mind that the spinal cord is considerably shorter than the vertebral column. The distance between the point of origin of the phrenic nerve at the fourth cervical segment and the sensory innervation of the uterus at the eleventh thoracic segments is only 10 inches (25 cm). This short distance is the margin of safety between the relief of pain and complete respiratory paralysis.

Local Anesthesia.—The local anesthetic drugs, used for infiltration of the perineum or for pudendal block, may cause serious reactions if given in large quantity or if injected into or rapidly absorbed into the vascular system. Reactions to these drugs may be either stimulation of the central nervous system, resulting in convulsions, or depression of the central nervous system, resulting in apnea and hypotension. The onset of these reactions may be immediate or insidious. The convulsions are treated by the intravenous administration of small quantities of soluble barbiturates. The hypotension is treated by the intravenous infusion of a dilute solution of a vasopressor drug. With either form of reaction efficient artificial respiration is the most important factor in the treatment, since in both instances severe asphyxia is prone to occur. Although pain is not completely relieved by local injection, this is probably the safest method of anesthesia.

Morbidity Due to Anesthesia.—All of the factors aforementioned, which may lead to a fatality, may also be a cause of morbidity if their degree of severity is limited. Since complications involving the respiratory and circulatory systems finally act by damage to vital organs, both mother and infant may show transient or permanent evidence of anoxia. Next in importance is aspiration pneumonitis, the frequency of which is not generally appreciated. As a cause of unexplained fever in

the postpartum period, aspiration pneumonitis and atelectasis must always be considered. Even with patients prepared for an elective operation regurgitation occurs in 14 per cent; in half of these, aspiration into the tracheobronchial tree results. If the induction of anesthesia is difficult, the regurgitation rate is 25 per cent.[1] If the patient leaves the delivery room with a clear tracheobronchial tree and has regained her ability to cough vigorously, the risk of atelectasis and pneumonia is almost entirely eliminated.

Nausea and vomiting following anesthesia are still troublesome in some instances. The use of drugs to prevent or to treat nausea and vomiting offers some promise, but no drug has yet been found that is both effective and safe for this purpose.

The importance of a lumbar or caudal injection as a cause of meningitis, epidural abscess or arachnoiditis is not sufficiently appreciated. The organisms involved usually are those that inhabit the skin or the perianal regions. Keefer[2] reported 14 cases of meningitis following spinal anethesia for delivery, and in all instances the causative organism was a gram-negative rod. Stanley[3] reported that in 78 per cent of the cases of meningitis the condition was due to Pseudomonas aeruginosa, a common skin bacterium, and followed lumbar puncture. In the induction of regional anesthesia the strictest aseptic technic is mandatory.

Trauma incident to spinal or caudal anesthesia produces backache due to hemorrhage into the tissues. In addition, there may be injury to the ligaments of the spine, the meninges, the periosteum, the veins, the nerve roots, the cauda equina, the spinal cord and the nucleus pulposus of the intervertebral disc. Laminectomy, performed to correct a herniated disc resulting from lumbar puncture, has been reported by many neurosurgeons. Trauma to the tissues of the central nervous system leads to irreversible degeneration. The backache that follows spinal anesthesia may be due not only to the anesthesia itself but to the stress of pregnancy or the improper manipulation of the legs in and out of the stirrups.

Neurologic complications following conduction anesthesia may be traumatic, infectious, neurotoxic or due to alterations in the spinal fluid dynamics. Myelitis and the cauda equina syndrome, with paralysis of the bladder and rectal sphincters, are occasionally encountered. Concentrated anesthetic drugs, contaminants or material mistakenly used as an anesthetic drug may destroy nerve tissue. Drugs used for conduction anesthesia should be sterilized by autoclaving, and the contents of the ampules should be rechecked at least once before being used.

Troublesome but not alarming is the frequent occurrence of spinal puncture headache, which has been reported as occurring in 25 per cent of all obstetric patients.[4] Signs of meningeal irritation are often present concurrently with the headache. There may also be paralysis of the cranial nerves, usually the abducens, as well as other visual disturbances, tinnitus and dizziness. These difficulties are caused by alterations in the spinal fluid dynamics that occur after spinal puncture. Their incidence may be reduced by using small-gauge needles. The treatment of this type of difficulty, if severe, is the injection of saline solution into the epidural or subarachnoid space in quantity sufficient to restore the spinal fluid pressure. Analgesics and occasionally narcotics may be needed to keep the patient comfortable. Many drugs have been recommended for the treatment of the postspinal headache, but none is uniformly successful.

Following inept venipunctures or inadequate care in withdrawing the needle, extravasation of blood from the vein may lead to a troublesome hematoma. The use

of concentrated solutions may cause phlebitis. The extravascular injection of irritant drugs, such as pentothal sodium, may result in severe pain, inflammation and sometimes sloughing of the tissues. Intra-arterial injections of such drugs may lead to gangrene and loss of the extremity.

Prevention of Anesthetic Complications.—In view of what has already been said of the causes of morbidity and mortality in obstetrical anesthesia, preventive measures can easily be understood. The patient should be properly prepared for the administration of anesthesia. The type of anesthesia used should be selected according to the condition of the patient. Before an anesthetic is administered, the equipment necessary should be assembled and in good working order. The equipment for suction and for the administration of oxygen must be present in every instance. With all forms of anesthesia, whether general, regional or local, the patient's respiration must be maintained and an adequate oxygen supply assured. Assisted respiration or artificial respiration with oxygen should be utilized as necessary. Hypotension should be prevented. Neurogenic hypotension is treated by the use of vasopressor drugs, and hemorrhagic shock is treated by the administration of blood or blood substitutes. Local anesthetic reactions may be prevented by the use of dilute solutions and small amounts. Intravascular injection can be prevented by repeated aspiration with the syringe before injection. Neurologic complications can be prevented by the use of small doses and dilute solutions of the anesthetic agents and by injecting them carefully and with aseptic technic.

Careful as one may be, difficulties may develop in individual patients as a result of the administration of these potent and toxic drugs. If the severity of the reaction is allowed to progress, or if it is unrecognized, it soon becomes difficult or impossible to restore the patient to normal. For this reason, one of the most important things is the constant observation of the patient and the vital signs. If an adequate record is kept, one may be able to note the beginning of difficulty by the trend of the record, before it has become serious. Failure to keep a good record usually means failure to make observations, and lack of observation invites disaster.

After the baby has been born and the episiotomy repaired, the nursing and medical staff find many chores to occupy their time. This is a period of great danger to the patient, whose physiologic systems have not yet returned to normal. She may bleed profusely, fall off the table, obstruct the airway or drown in vomitus. A trained person must be in constant attendance until the patient has regained stability and control of her respiratory and circulatory systems.

Many obstetricians and many surgeons ask the anesthetist to suggest a safe routine or a safe agent to be used for all patients. It is impossible to comply with this request. Each patient must be treated individually, with the aim of using a minimum amount of the least toxic agent that can accomplish the desired result. One does not drive a tack with a sledge hammer, nor does one use a mallet as a pile driver.

The Critical Factor.—Much has been said in other papers as well as this about advantages, disadvantages, hazards, protective mechanisms and other items related to the safety of anesthesia. The most important point we have left for last: a competent anesthetist is the *sine qua non* of safe anesthesia. No drug and no technic is any safer than the person who administers it. Safety in obstetric as well as in surgical anesthesia is to be achieved not through a search for different technics or new drugs but through the training and the

availability of competent persons to administer the anesthetic. The senior men in the medical profession, surgeons, obstetricians, internists and men of all specialties must recognize the danger and encourage young physicians to take training in anesthesia, so that they may be the means of providing safe anethesia for the community.

SUMMARY

As other causes of maternal mortality are conquered, anesthetic deaths assume an increasingly important role. Most anesthetic deaths are preventable. They result from derangements of the circulatory and respiratory systems and must be anticipated, prevented or promptly treated. Asphyxia due to vomitus, the most frequent cause of preventable anesthetic death, can be prevented if the obstetrician instructs the patient to avoid eating after labor begins and if general anesthesia is not employed after the patient has eaten. The most important factor in safe anesthesia is not a particular drug or method but the competence of the anesthetist.

RESUMEN

A medida que las causas de mortalidad materna van siendo vencidas adquieren una mayor importancia las muertes anestésicas. La mayor parte de las muertes anestésicas son evitables. Estas muertes son motivadas por alteraciones en los sistemas circulatorio y respiratorio y deber ser diagnosticadas en su comienzo, prevenidas y tratadas a tiempo. La axfisia producida por vómitos es la causa más frecuente de muerte anestésica, y puede evitarse si el tocólogo instruye a la paciente para que no tome hada una vez que el trabajo del parto comienza; como norma no se debe emplear anestesia general si ha comido ya algo. El factor más importante para la anestesia sin peligro no es un anestésico o un método determinado, sino la competencia del anestesista.

RIASSUNTO

Le morti in corso di anestesia, fra le altre forme di mortalità materna, divengono sempre più frequenti. Molte di esse potrebbero essere evitate, o prontamente curate. L'asfissia secondaria al vomito, che è la più frequente causa delle morti da anestesia evitabili, può essere prevenuta se l'ostetrico avvisa la paziente di non bere più dopo l'inizio del travaglio, o se non pratica la narcosi in pazienti che abbiano bevuto. Il fattore più importante ai fini di una anestesia sicura non è un medicamento o un metodo particolare, ma la competenza dell'anestesista.

ZUSAMMENFASSUNG

Während Ursachen für die Sterblichkeit der Mütter bei der Entbindung unter Kontrolle gebracht werden, treten die Todesfälle infolge von Narkose mehr in den Vordergrund. Die meisten Narkosetodesfälle lassen sich verhüten. Sie sind die Folge von Störungen des Kreislaufs oder der Atmungsorgane und müssen vorausgesehen, vermieden oder unverzüglich behandelt werden. Die häufigste Ursache des vermeidbaren Narkosetodes ist Erstikkung infolge von Erbrechen. Dies lässt sich dadurch vermeiden, dass der Geburtshelfer der Patientin die Nahrungsaufnahme nach Einsetzen der Wehen verbietet oder aber durch Ablehnung einer allgemeinen Narkose, wenn die Patientin bereits gegessen hat. Die wichtigste Rolle bei der Durchführung einer gefahrlosen Narkose spielt nicht die Art des angewendeten Medikaments oder der Methode sondern die Erfahrenheit des Anaesthesiologen.

RÉSUMÉ

Alors que de nombreuses causes de mortalité maternelle lors de l'accouchement ont pu être vaincues, les dècès par anesthésie ont pris une importance plus grande. La plupart pourraient cependant être évités. Lls résultent de dérèglements des systémes circulatoire et respiratoire, qui doivent être prévus, évités, ou très rapidement traités. L'asphyxie due aux vomissements est une des causes de décès les plus fréquentes durant l'anesthésie. L'obstétricien devrait toujours avertir les patientes de s'abstenir de manger dès le travail commencé (ne jamais pratiquer d'anesthésie générale après un repas). Le facteur de sécurité le plus important ne réside pas dans le produit employé mais bien dans la compétence de l'anesthésiste.

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Medicine has become a group of professions rather than a single one. The many committees which have considered the curriculum during recent years have all been harassed by this increasing complexity; on the one hand by the demand of fresh subjects as radiology, psychological medicine, and manipulative surgery for admission, on the other by the clear desirability of shortening the whole course, so that it shall encroach less rather than more on the school years and allow a continuance of that general education which is of paramount importance to the future doctor.

—*Ogilvie*

The Weight Stream in Charcot's Disease of Joints

Charcot's Disease of the Foot and Ankle; Comment on Spontaneous Fracture of the Heel Bone and on Posterior Extra-Articular Fusion of the Ankle

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CHARCOT'S disease is the sequel of the physiologic weight force on a joint without proprioception. The destruction of a joint in a case of Charcot's disease, and the succession of events in destruction, are not haphazard but follow certain rules, of which the most important is the rule of shear. The biologic reaction of a joint subject to unfelt torsional stresses—hyperplastic periostitis of its shaft bones, new bone in the joint capsule, bony detritus in the synovial membrane, large fragment sequestration of bone, and unregulated and excessive callus in fracture—does not stay the illness. The illness is stayed when the joint ceases to exist as a functioning entity by fusion *within* or *without* the joint. The latter is preferable. Or, the joint must be reconstructed to break up the vicious circle of destruction that causes deformity and, in turn, further destruction and further deformity, and so on. And yet, unfortunately, the reconstructed joint lacks proprioception, so that it too may become neuropathic unless it is protected against the adverse effect of shear.

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The two sites of this illness in the foot and ankle are linked by the astragalus, which is the bridge joining them. Charcot's disease of the foot begins in or near the head of the astragalus and soon involves the astragaloscaphoid joint. (Milgram classified Charcot's disease of the ankle into four types: (a) primary destruction of the ankle joint, sometimes initiated by a painless Pott's fracture; (b) primary destruction of the subastragaloid joint in which the astragalar head melts away; (c) destruction of the midtarsal joint (most frequent type), and (d) destruction of the joints between the metatarsal bones and the cuboid and cuneiform bones.)

Steindler stated that the ankle was involved in 17.7 per cent of Charcot joints and that there was significant association with similar disease of the knee joint. He treated 33 Charcot ankle joints conservatively. Of 2 operative cases, he did a subastragaloid fusion in 1 and achieved good result; in the other, he performed an osteotomy of the internal malleolus to correct varus of the ankle. Cleveland reported successful fusion of a Charcot

ankle joint in 1 case.

Mechanics of Destruction in Charcot's Disease of the Ankle and Foot.—The vectorially vertical weight force passes through the body of the astragalus to the ground in the normal foot. It is opposed by the collinear but opposite and equal counterweight force. The obliquity of the astragalus directs the major oblique component of the weight force along the long arch of the foot. The minor oblique component, perpendicular and coplanar to it, passes through the heel bone. The inferior calcaneoscaphoid ligament, which cups the head of the astragalus, resists the downward and medial descent of the head. Since the weight force passes through the center of the body of the astragalus, there is no moment of rotation.

Only after some destruction of the astragalar head or the subastragaloid joint does the weight force pass eccentrically to the center of the body of the astragalus. The differential elements of the weight force do not delineate a force infarct as they do in the hemispherical hip joint, for the superior surface of the bone that articulates with the tibia is convex from before backward and slightly concave from side to side (a surface of negative curvature). The effect of these elements is divergent on a concave surface and convergent on a convex surface, the total effect converting the astragalus into an amorphous mass of condensed bone (Flori and Chaumont).

The eccentric position of the weight force and tilt must result in shear and, sooner or later, a shear fracture of the astragalus and even of the body of the scaphoid bone.

The angle of shear, which shears the astragalus through its neck or body, is the angle of adduction and inversion of the foot. If A is the cross-sectional area of the astragalus, then the shear of the weight force is $\frac{W \sin\phi}{A} \frac{\text{lb.}}{\text{inches}^2}$, where ϕ is the angle made by the astragalus with the horizontal and W is the weight force. The direct stress is $\frac{W \cos\phi}{A} \frac{\text{lb.}}{\text{inches}^2}$.

Unchecked, the subastragaloid joint and midtarsal joints are soon destroyed. The ankle joint itself is secondarily involved. An abnormal strain is thrust upon the ankle joint by the deviated (often inverted) foot. This induces shearing stress in the tibia and fibula, expressed as parallel periostitis. New bone forms in the common capsule that envelops the posterior part of the subastragaloid and the ankle joint. Roentgenograms of the Charcot ankle show a final step of consequential displacement and rotation of the articular part of the astragalus that enters the mortise of the ankle joint. *All the component joints of the astragalus become destroyed in this disease.* The distinction between primary and secondary Charcot's disease of the ankle becomes academic when the disease is advanced.

The destructive effect of eccentric movement is spent in the back part of the foot, for the weight force is not deflected to the fore part. There is a non-singular lack of association between Charcot's disease of the back part of the foot and perforating ulcer occurring as the sequel of neuropathic disease of the metatarsophalangeal and interphalangeal joints.

Surgical Treatment of Charcot's Disease of the Foot and Ankle. — Surgical therapy should be based on realization of the sequence of events in destruction and should use this sequence, especially with early disease. The fusion of the component joints of the long arch of the foot, especially the astragaloscaphoid joint, may check Charcot's disease if the two favorable results of this fusion follow—no motion in the subastragaloid joint and *restricted* motion in the ankle joint.

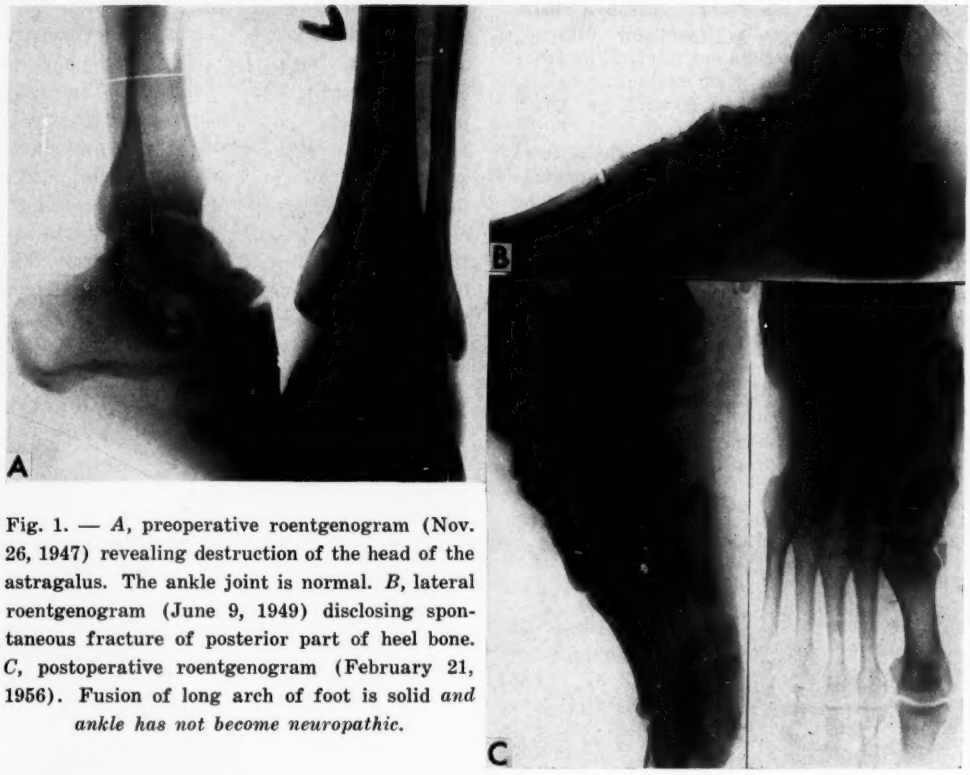

Fig. 1. — *A*, preoperative roentgenogram (Nov. 26, 1947) revealing destruction of the head of the astragalus. The ankle joint is normal. *B*, lateral roentgenogram (June 9, 1949) disclosing spontaneous fracture of posterior part of heel bone. *C*, postoperative roentgenogram (February 21, 1956). Fusion of long arch of foot is solid *and ankle has not become neuropathic.*

CASE 1 (*Charcot's disease of the hip; the development of early Charcot's disease of the foot, successfully treated; follow-up of more than seven years*).—In December 1946 a man 42 years old underwent an ischiofemoral (Brittain) osteotomy fusion of a Charcot hip (the right). This operation resulted in fusion in the three principal planes. The follow-up time has been more than nine years. In November 1948 the left foot became painful and swollen. A collar-like swelling overlay the astragalar head. Ankle joint motion was free; subastragaloid joint motion was a little restricted. The tender astragalar head descended medially on weight bearing.

Roentgenograms disclosed a destructive process limited by a sclerotic border in the astragalar head (Fig. 1*A*). The head was compressed. The joint between the astragalus and the scaphoid showed no significant change.

The diagnosis of Charcot's disease of the astragalar head was made, and intra-articular and extra-articular fusion of the longitudinal arch were done on December 18, several countersunk iliac bone grafts spanning the astragaloscaphoid and scaphoid-cuneiform joints.

An oval, dime-sized ulcer, filled with yellow-brown granulation tissue, was observed in the astragalar neck just behind the head. The cartilage of the astragalar head was more opaque than normal. The joints between the astragalus, and the scaphoid and cuneiform bones, contained thick synovial fluid.

One tissue section showed "some evidences of periosteal new bone deposition" and another a focus of subchondral scarring. There were no striking changes in the articular cartilage or the synovium.

The plaster was removed on April 8, 1949, and the heel became painful soon after the patient began walking. The existence of a spontaneous avulsion fracture of the heel bone by pull of the achilles tendon on the atrophic

bone (Fig. 1 *B*) was recognized on June 8. The Charcot osteopathic condition did not enter the stiff subastragaloid joint. This fracture healed well, but with exuberant callus, under simple immobilization with an Unna paste bandage.

The operative fusion has served the patient well. On Feb. 21, 1956, the non-neuropathic left ankle moved from 90 to 110 degrees. The long arch of the foot remained fused, with no motion in the subastragaloid joint. The fore part of the foot was abducted a little. The posterior tuberosity of the heel was broadened. The patient could walk any distance. The follow-up covered more than seven years (Fig. 1 *C*).

This early form of Charcot's disease, expressed as a juxta-articular erosion of the head of the astragalus, would soon have entered its joint with the scaphoid.

Comment on Spontaneous Fracture in Charcot's Disease.—Spontaneous fracture occurs less frequently in tabetic patients than does arthropathic change. The bony processes and apophyses, subject to muscle pull, are usually affected. Normal bone knows how to respond to muscle power and can resist untoward demands. Charcot bone does not, for it lacks proprioception. (Corbin and Hinsey noted that deafferentation results in loss of the protection afforded by the proprioceptive mechanism.) Syphilitic osteitis weakens the bone so that muscle pull or weight compression can break it. Fragmentation of the patella is probably due to quadriceps pull.

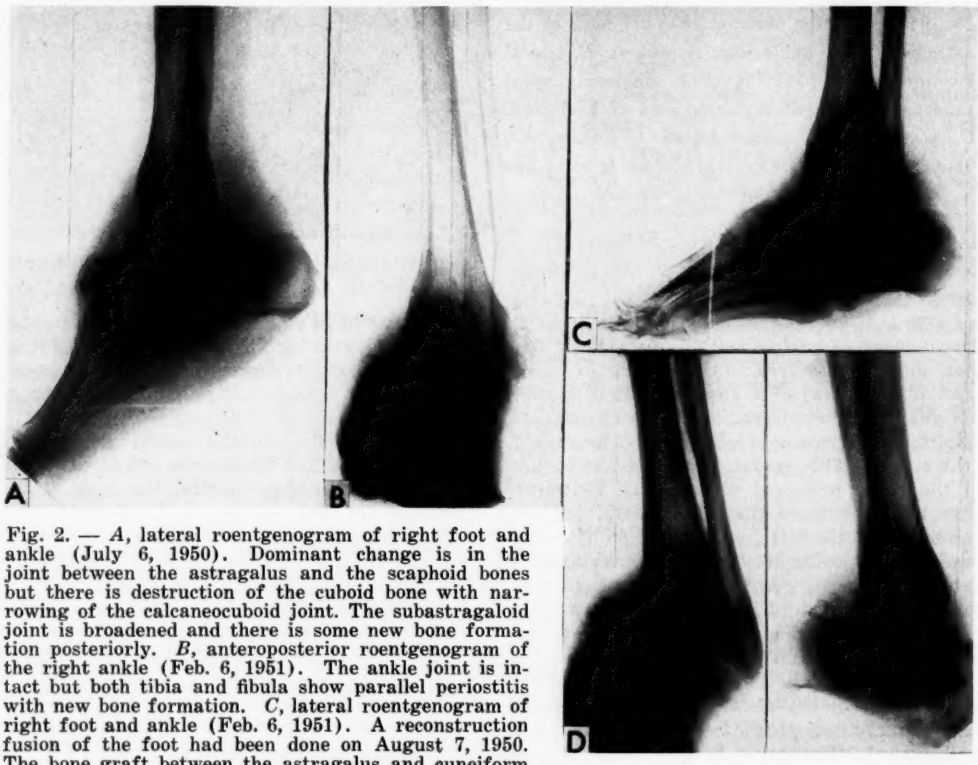

Fig. 2. — *A*, lateral roentgenogram of right foot and ankle (July 6, 1950). Dominant change is in the joint between the astragalus and the scaphoid bones but there is destruction of the cuboid bone with narrowing of the calcaneocuboid joint. The subastragaloid joint is broadened and there is some new bone formation posteriorly. *B*, anteroposterior roentgenogram of the right ankle (Feb. 6, 1951). The ankle joint is intact but both tibia and fibula show parallel periostitis with new bone formation. *C*, lateral roentgenogram of right foot and ankle (Feb. 6, 1951). A reconstruction fusion of the foot had been done on August 7, 1950. The bone graft between the astragalus and cuneiform is seen. *D*, anteroposterior and lateral roentgenograms of April 17, 1952, reveal a progressive breakdown of the astragalus and now great involvement of the ankle joint. The foot is secondarily luxated into varus.

Fig. 3.—Extensive neuropathic disintegration of the right astragalus with extension of disease into each of its joints. The primary lesion was probably in the astragaloscaphoid joint. Note the excess periosteal callus about the fractured left tibia overlying the vitallium plate. There is synostosis between the tibia and the fibula.

A spontaneous fracture of the posterior apophysis of the heel bone was a complication in this case. Schwede called it a tabetic osteopathic condition, not a spontaneous fracture. Both Schwede and Laignel-Lavastine described a red swelling at the site of fracture that looked like a phlegmon. Despontin and Fernando Corsellas expressed the opinion that this fracture followed loss of ossein, the organic substance of bone, and that such fractures are most frequent in the ataxic stage. Castay, however, considered this an early and leading sign of tabes.

This vague concept of cause was made more specific by Capener, who found the cause of fracture to be syphilitic osteitis. Sections of the heel bone revealed vascular granulation tissue filling the marrow spaces, causing necrosis and absorption of bone.

The ilium is less often a site of spontaneous fracture (Weber). Other bones may break, too. Roger and Arnaud described the case of a woman 65 years old, who had been tabetic since the age of 48. In 1928 she broke two right metatarsal bones. In 1931 three ribs showed spontaneous fractures, as did the spinous processes of some cervical vertebrae. In 1935 the right elbow broke, and in 1936 the lower third of the right forearm.

CASE 2 (*a less happy result after reconstruction and fusion of a foot, greatly destroyed by Charcot's disease; consequent involvement of the ankle joint*).—A woman 26 years old had Charcot's disease of the right foot, secondary to congenital syphilis. A lateral roentgenogram taken on July 6, 1950,

Fig. 4.—Roentgenograms (April 13, 1948) of a fused Charcot ankle and foot after operation and secondary infection. The soft tissues were thickened, indurated and ulcerated, especially over the lower part of the tibia.

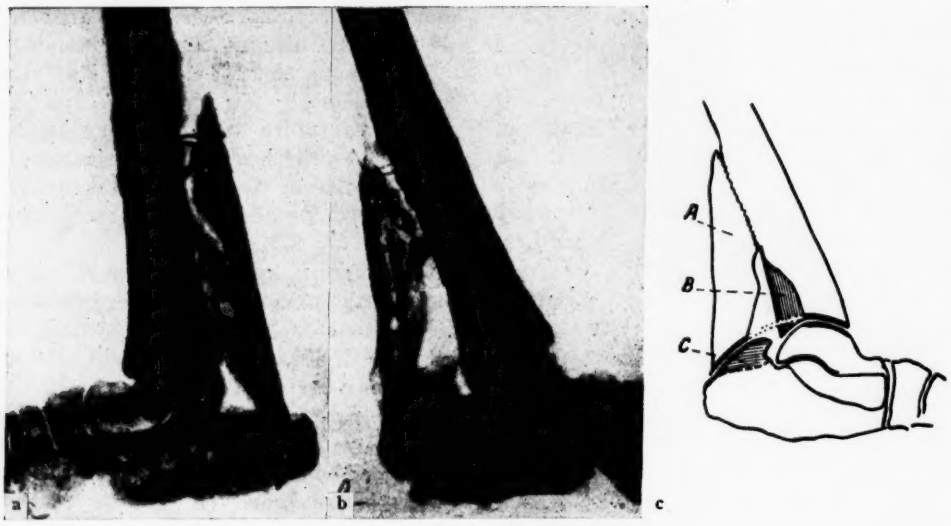

Fig. 5.—Illustration from page 83 of W. Muller's book (1924).

showed the extent of change (Fig. 2 *A*). The ankle joint seemed free of this disease, even though there was some new bone formation in the region of the posterior tubercle of the astragalus. Fusion of the foot was done on August 7. Through the lateral (Kocher) incision great destruction of the subastragaloid joint was revealed with sequestration of the dorsal half of the cuboid bone plus other fragments of bone. The short neck of the astragalus was removed. Combined subastragaloid and calcaneocuboid joint fusion was done through the lateral incision. The second or medial incision extended from the tip of the internal malleolus to the midpart of the first metatarsal bone. The soft tissues were edematous. The loose first cuneiform bone was excised. The dorsally displaced scaphoid bone, which had been rotated so that its long axis ran from dorsal to plantar, was excised. The excision of all, or nearly all, necrotic and sclerotic bone effectively débrided the midtarsal area. The foot was neutrally held, and an iliac graft was countersunk in the gutter that extended from the astragalus to the shaft of the first metatarsal bone.

The plaster was changed on August 21; both wounds had healed. A new plaster was applied to the knee. Clinically, on October 26, when this plaster was removed, *the back part of the foot seemed fused.* A walking plaster was put on, and later the patient was given a brace and high shoe (Fig. 2, *B* and *C*). My note of April 26, 1951, stated that the fusion had failed. The last follow-up examination (April 17, 1952) indicated advance of destruction, with severe involvement of the ankle joint. The inverted foot was medially displaced (Fig. 2 *D*).

The secondary involvement of the ankle joint is likewise visible in the comparable roentgenograms of Figure 3, which show great destruction of the astragalus and extension of the disease into each of its component joints. The ankle joint is extensively involved.

CASE 3 (*fusion of the Charcot foot and ankle, probably by infection after an operation to fuse the ankle by bone grafting; below-knee amputation later*).—In 1936 a 43-year-old Negro fell 5 feet sustaining a fractured right ankle. A bone graft operation to fuse the ankle was done in 1940. The patient was well for three years, after which "loose pieces of bone" were discharged. A second bone graft operation was done in 1943, but the wound broke down. Many pieces of bone were removed in 1945, and the limb was encased in plaster for six months. The patient used crutches for one year. (All these operations had been done elsewhere.) He had done little or no work for ten years.

A large sequestrum of bone was removed in the outpatient department on April 6, 1948, from a deep crevice on the anteromedial part

of the foot just below the ankle. The sequestrum, which measured 1.25 cm., was covered with adherent granulation tissue.

The right ankle and the back part of the foot were swollen. The skin over the lower half of the leg and foot was thickened, scaly and depigmented in part. An oval ulcer 2 inches (5 cm.) long and 1 inch (2.5 cm.) wide exposed the anterior face of the tibia 3 inches (7.5 cm.) above the ankle. A probe passed into the deep crevice from which the sequestrum had been removed struck bone. Pus was discharged from this opening. The ankle joint and the subastragaloid and midtarsal joints were solidly fused. The toes were clawed. The patient limped badly.

Roentgenograms (Fig. 4) disclosed fusion of the ankle and the subastragaloid and midtarsal joints by dense sclerotic bone. There was extensive irregular periostitis of the lower ends of the fibula and tibia. A large, triangular free bone fragment was seen near the scaphoid. The anterior part of the fifth metatarsal bone was absent.

A right mid-leg amputation was done on July 26. The friability of the necrotic soft tissues made ligation of the main vessels most troublesome after the amputation. The postoperative course was good.

It is not certain whether the bone graft operations or the secondary infection fused the ankle and the foot to yield, in effect, a panastragaloid arthrodesis; more probably it was the infection. It is certain that the prolonged infection and necrosis of the soft tissues and bone made amputation necessary despite the solidity of the fusion.

The ankle joint seems not to be a primary site of Charcot's disease; it is secondarily involved by increasing destruction of the astragalus. The articular part of the astragalus which enters the mortise of the ankle joint seems last affected. Failure to fuse the foot extends the neuropathic condition into the ankle joint proper by failure to achieve rigid immobilization of the astragalus.

Any therapy must take this fact into due consideration. There are, then, three operative solutions for advanced disease, apart from amputation.

(a) fusion of the component joints of the astragalus, creating, in effect, a panastragaloid arthrodesis; (b) a reconstruction-fusion operation on the foot, to which is added posterior extra-articular fusion of the ankle joint, or (c) astragalectomy, to which should be added posterior extra-articular fusion of the ankle. Nature itself tries to achieve an astragalectomy for natural cure. Yet astragalectomy as the solution in itself is improper for the new ankle joint is without proprioception.

Leger and Boreau surveyed the surgery of Charcot joints, reviewing in particular the French literature. Only 3 cases of operation on the Charcot ankle had been recorded since the report of Jaboulay in 1904. In all 3, astragalectomy had to be done because of bony necrosis with infection. The infection healed quickly and several years later there was solid fusion of

Fig. 6.—Lateral roentgenogram taken May 27, 1948.

the ankle, probably consequent upon the infection.

Leger and Boreau themselves reported an example of Charcot's disease of both ankles in a woman aged 64. Astragalectomy of the right ankle was done on Oct. 15, 1943, and in February, 1944, there was little motion in the ankle, which the patient used well. In the left ankle the disease had advanced.

Fusion of the astragalectomized ankle by a nail driven through the heel into the tibia may or may not succeed.* The experiences of Stack and Lichtenstein, who used *one* intramedullary nail to fuse the knee, predicate failure if torsional stresses exist after the operation.

Probably the best combination is astragalectomy plus posterior extra-articular fusion of the ankle. (It is generally true that intra-articular fusion of a Charcot joint fails, the hip joint and the knee joint being fine examples of this.) I have not had the opportunity to treat advanced Charcot's disease of the ankle by this method. I have used the method of posterior extra-articular fusion of the ankle in 2 cases for illness other than Charcot's disease.

Comment on Posterior Extra-Articular Fusion of the Ankle.—This operation was derived from my experience with the Brittain fusion for Charcot's disease of the hip which convinced me that a short extra-articular perpendicular graft under compression resulted in fusion where fusion was ordinarily most difficult to achieve. Use of the principle elsewhere seemed to have merit, especially in the ankle.

In the first of my 2 cases operation was performed Feb. 16, 1948. Müller's experimental attempt to achieve posterior fusion of the ankle in the leg of the dog ended in a false joint distally. The line of the new joint between the graft and the heel bone became the posterior extension of the ankle joint proper (Fig. 5, taken from Müller, 1924). In Campbell's technic of posterior fusion for tuberculosis of the ankle joint (1949), leaves of tibial bone turned down were imbricated with leaves of calcaneal bone turned up. Staples (1956) described a technic for posterior fusion of the subastragaloid and ankle joints, which he may have used since 1949 or 1950 (his case reports give no exact dates). In his opinion, the proper indication for this operation is combined disease or injury of the subastragaloid and ankle joints. He employed the operation in treating 3 patients with combined fracture of these two joints and 1 with tuberculosis of the ankle joint. He performed it also in 2 cases for relief of pain after astragalectomy.

Mechanics of This Operation.—The short graft extends between the tibia and the heel bone. If fusion takes place, both the subastragaloid and the ankle joint will become fixed. The graft is perpendicular to the heel bone, or almost so, when it is made to lie flush with the posterior surface of the tibia, the foot being in equinus. This may demand resection of the posterior malleolus of the tibia and the posterior tubercle of the astragalus. In any other position the graft is oblique, its obliquity increasing the more posteriorly the heel slot in which it rests is made. The technic of proximal fixation of the graft varies directly with the acuteness of the angle of the graft. An oblique graft can be passed through a tibial slot; the perpendicular graft can only be screwed or nailed against the bone-raw surface of the tibia. Screw fixation of the distal part of

*It is possible to fuse the ankle joint (and probably any joint) by approaches from the medial side (White and Hoyt) or the lateral side (Adams, Horwitz), from behind or in front, or by the passage of an intramedullary bone graft driven from the tibia through the astragalus and into the heel bone (Hyatt, Howard Rosen), or through the heel into the tibia by the technic of Bingold. (I had once seen Telson do an operation like Bingold's many years ago.)

In none of these approaches and in the technics made necessary by the approach chosen can any claim other than ingenuity be made. It is the principle of fusion itself which is the original idea. A search of the literature is sometimes chastening but always revealing. Thus, Ducroquet and Lescoeur in 1949 reported on posterior extra-articular fusion of the ankle, combining it with anterior fusion or an intramedullary bone graft driven through the heel into the tibia. The use of a Smith-Petersen nail instead of the bone graft was also suggested.

the graft is not needed. Grafts that are not too oblique will result in rapid and solid fusion by salutary weight compression, the graft seeking to some degree the security of perpendicularity.

The length of the unburied part of the graft should not exceed 3 to 4 inches (7.5 to 10 cm.), and its width should be 1 inch (2.5 cm.) or more. If the graft is too long and too narrow, it will break in its middle or a false joint will form at one end or the other, as in the experimental animal described by Müller. The distal false joint becomes paired with the true ankle joint to provide directional constraint of motion and hence some measure of fusion.

Since the graft fuses the ankle and subastragaloid joints simultaneously, it is advantageous when there is *simultaneous* disease of these two joints, as in cases of fracture deformity or in the equivalent situation of the painful astragalectomized foot. It is equally valuable in a situation characterized by *consecutive* disease, as in Charcot's disease or tuberculosis of the ankle. A somewhat oblique graft will have to be used where the anatomic situation demands true extra-articular fusion. Otherwise, the technic of Staples is easier and mechanically more secure.

Fig. 8.—Lateral roentgenogram taken May 23, 1950. The ankle was well fused, but tuberculosis was still active.

Fig. 7.—Lateral roentgenogram taken March 1, 1949.

CASE 4 (*posterior fusion in combined tuberculosis of the ankle and subastragaloid joints*). —A man aged 23, a Puerto Rican, had proved tuberculosis of these joints, which apparently began when he was 8 years old. The illness was not relieved by immobilization of the limb in plaster, with *steptomycin daily* from December 23, 1947, until he had taken 56 Gr.; dizziness and tinnitus made it mandatory to discontinue the drug whose obvious effect was the rapid healing of an ulcer over the external malleolus.

Despite the complication of a cold abscess over the right fourth costochondral junction, operation was done bloodlessly, with use of

Fig. 9.—Lateral roentgenogram taken Nov. 10, 1954.

the pneumatic tourniquet, on Feb. 16, 1948. The tendo Achilles was exposed through a long medial incision. It was divided just above its insertion and reflected proximally. The posterior lip of the tibia was removed. Aside from this, the operation was extra-articular. A bone graft was taken from the anterior surface of the same tibia and its proximal end beveled to allow for its passage through a transverse slot in the posterior surface of the distal fifth of the tibia. The distal end of the graft was driven into a transverse slot in the superior plateau of the heel bone. The sharp proximal end of the graft was brought into the tibial slot and the graft held firmly when the foot was put into slight equinus. The cancellous surface of the graft faced the rawed surface of the tibia; cancellous bone was packed into the triangular interspace in front of the graft. A long leg plaster was put on after suture of the tendo Achilles and closure of the two operative wounds.

Under combined penicillin and streptomycin therapy, the patient ran a gentle postoperative course.

The ankle joint seemed well fused when the plaster was removed on May 27, 1948 (Fig. 6). The wound was healed except at one point, where there was a sinus. The new plaster was removed on June 29, and several sinuses were observed about the ankle. My note of March 1, 1949, stated that the fusion of the still warm ankle was solid and that there were no sinuses. The patient had been given streptomycin therapy as an in-patient in two of the City's hospitals, the last hospitalization being early in December, 1948. The chest wall now showed neither abscess nor ulcer. Yet again on March 15, 1949 (Fig. 7) there were two sinuses in the ankle and a recurrence of the abscess of the chest wall. The patient walked with two crutches and put little weight on the foot. Despite the solid fusion of the ankle, the sinuses came and went and were still being dressed when he last visited the Clinic, on June 1, 1950 (Fig. 8). The fusion did not halt the activity of the disease. This patient has been lost to our Follow-up Clinic.

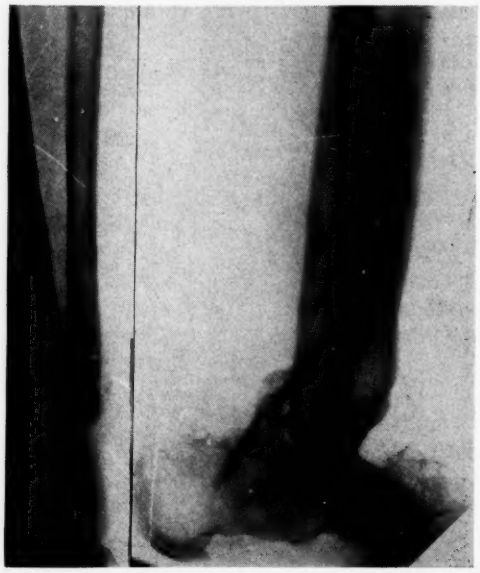

Fig. 10.—Lateral roentgenogram taken April 18, 1956, revealing resorption of the tibial slot of the graft. It is possible that the middle part of the graft, about which there is a good deal of new bone, may have broken to indicate that the graft was too long.

CASE 5 (*osteomyelitis of the astragalus consequent on compound fracture of the ankle; removal of the astragalus; posterior extra-articular fusion of the ankle*).—A man 59 years old, sustained a compound fracture-dislocation of the right ankle on April 18, 1952, in an automobile accident. The internal malleolus and the lower end of the tibia had been resected for sequestration some time before his admission to the Hospital for Joint Diseases on September 12, 1952. There was a fungating wound on the inner side of the ankle. The right foot was thrust into varus by the long fibula.

The necrotic astragalus was removed on September 22. The posterior tibial tendon, which was torn and necrotic, was resected. The long fibula had to be shortened level with the tibial stump, which was made wedge-shaped and set into a matching slot in the foot. The foot was set back as in the Whitman astragalectomy and held in valgus and equinus. It was hoped that fusion would take place. The wound healed well.

The plaster was removed near the end of February, 1953. The patient was given first a brace and later a molded leather ankle support. He continued to walk with pain, even with the aid of two crutches. Because of continued pain, a posterior extra-articular fusion of the ankle was done on July 22, 1954, through an incision placed laterally to the heel cord. The tendon was freed so that it could be mobilized. The fibula lay 1 inch (2.5 cm.) back of the tibia. The deep muscles of the leg were brown and atrophic. The fat was edematous. The atrophic heel bone cut easily with the knife when the transverse groove was made in it. The slot in the tibia was placed 1½ inches (3.8 cm.) above the ankle joint. The superior surface of the heel bone and the posterior surface of the tibia were made raw with the chisel. A bone graft 4 to 5 inches (10 to 12.5 in.) long and the width of the tibia was taken from the upper and anterior aspect of the same tibia. The graft was passed deeply into the heel slot, and then its sharp proximal end was implanted into the tibial slot. When the foot was plantarly flexed the graft fitted tightly. The marrow bone of the graft was placed in the triangular interspace in front of the graft.

There was solid fusion on September 7, when the long plaster was removed and a short walking plaster applied. On December 1 (Fig. 9) the patient used one crutch and a cane. He wore an elastic bandage and an elevated shoe with a heel lift of 2½ inches (6.3 cm.).

Fig. 11.—Advanced Charcot disease of the ankle for which midthigh amputation was done. Compare this with Figures 2*A* and 3. Note again the disruption of the subastragaloid joint and the great swelling of the posterior part of this joint and the ankle joint in whose combined capsule there is new bone formation. There is a great new bone formation between the tibia and the cuneiform bones dorsally. Most of the astragalus has been destroyed and the part which enters the mortise of the ankle is the largest survivor.

He was seen again on April 18, 1956, at which time he walked with two canes and an elevated shoe. About 2 to 3 degrees of motion existed in the ankle, and vague diffuse tenderness and pain were present about it. A lateral roentgenogram (Fig. 10) disclosed graft resorption at the tibial slot and fusion of the distal end of the graft to the heel bone. His condition was unchanged on May 29, 1957, but it was observed roentgenographically that the middle part of the graft had broken and healed, with abundant callus, in slight posterior angulation.

The final solution is ablation, below the knee if the knee is sound, and above it if it is not.

CASE 6 (*advanced Charcot's disease of the right ankle, mid thigh amputation*).—A white man 63 years old sprained his right ankle in 1943 while shoveling coal. In time this ankle became swollen and unstable. Roentgenograms (Fig. 11) revealed gross destruction of the ankle and the back part of the foot. A midthigh amputation had to be done on February 1, 1946, because of ascending infection of the foot and leg. In August, 1946, the patient was given an artificial limb, which he is still using.

SUMMARY

In the author's opinion, Charcot's disease of the ankle is secondary to neuropathic destruction that begins in or about the head of the astragalus. If the astragalus is destroyed in this disease, then it should be that adequate fixation of the astragalus will stay destruction. And this can be the only secret of cure. In the early stages, fusion of the long arch of the foot is successful. In the more advanced stages three procedures are possible: (a) panastragaloid arthrodesis, which fuses the component joints into which the astragalus enters; (b) fusion of the foot combined with posterior extra-articular fusion, or (c) astragalectomy with similar fusion. Astragalectomy by itself is improper, since the ankle joint lacks proprioception. Amputation is the measure of last resort in cases of great destruction of the component joints of the astragalus.

Comment is made on spontaneous avulsion fracture of the heel bone, a rare tabetic event and a sequel of a mechanical demand made on a bone that is insufficient because of syphilic osteitis.

ZUSAMMENFASSUNG

Der Verfasser ist der Ansicht, dass die Charcotsche Erkrankung des Sprunggelenks die Folge einer neuropathischen Zerstörung ist, die im oder in der Gegend des Kopfes des Sprungbeins ihren Anfang nimmt. In den frühen Stadien führt die Fusion des langen Fussgewölbes zum Erfolg. Für weiter fortgeschrittene Fälle gibt es drei Möglichkeiten der Behandlung: a) Arthrodese des gesamten Sprungbeins, wobei sämtliche Gelenke, an denen das Sprungbein beteiligt ist, verschmolzen werden; b) Fusion des Fusses, kombiniert mit extraartikulärer Verschmelzung und c) Resektion des Sprungbeins mit einer ähnlichen Form der Fusion. Die Sprungbeinresektion allein ist ein untaugliches Verfahren, da das Sprunggelenk keinen Muskelsinn hat. Die Amputation bildet die letzte Zuflucht in Fällen mit umfangreicher Zerstörung der Gelenke, an denen das Sprungbein beteiligt ist.

Es wird auf spontane Abrissfrakturen des Fersenbeins eingegangen, wobei es sich um eine seltene tabische Komplikation und um eine Folge mechanischer Beanspruchung eines infolge syphilitischer Ostitis untauglichen Knochens handelt.

RIASSUNTO

Secondo l'autore la malattia di Charcot della caviglia è secondaria alla distruzione neuropatica che si inizia in corrispondenza o in prossimità della testa dell'astragalo. Nei primi stadi è efficace la fusione dell'arco maggiore del piede. Negli stadi più avanzati vi sono tre possibilità: l'artrodesi panastragalica, che fonde tutte le articolazioni in rapporto con l'astragalo; la fusione del piede combinata con la fusione extra-articolare; l'astragalectomia combinata con la fusione. L'astragalectomia da sola è inefficace; l'amputazione è l'ultimo rimedio nei casi di distruzione estesa delle parti articolari dell'astragalo.

Viene trattata la frattura spontanea del calcagno che si verifica di rado nei tabetici come conseguenza di uno sforzo eccessivo eseguito su di un osso insufficiente per l'osteite sifilitica.

RESUMEN

Según la opinión del autor, la enfermedad de Charcot del tobillo es secundaria a la destrucción neuropática que comienza en, o al rededor de la cabeza del astrágalo. En los estadíos primarios de la enfermedad la fusión del arco largo del pié va seguida de éxito. En los casos mas avanzados existen tres posibilidades: (a) artrodesis panastragalina que funde todo el componente articular que rodéa al astrágalo; (b) la fusión del pié combinada con la fusión

extraarticular; (c) la astragalectomía con fusión similar. La astragalectomía simple es inadecuada pués hace perder la propiocepción de la articulación del tobillo. La amputación es un último recurso para los casos de gran destruccuín del componente articular del astrágalo. Se comenta el caso de una fractura por avulsión del calcáneo, cosa excepcional, en un tabético a consecuencia de exigencia de un trabajo excesivo a un hueso insuficiente a causa de una osteitis sifilítica.

SUMARIO

Na opinião do autor, a enfermidade de Charcot do tornozelo é secundária a destruição neuropática que se origina na ou cêrca da cabeça do astragalo. Nás fases primárias do mal a fusão do arco longo do pé é bem sucedida. Nas fases mais avancadas, ha três procedimentos exequiveis. (a) A artrodese pan-astragaloide que funde todas as juntas em que torma parte o astrágalo. (b) Artrodese do pé com fusão extra articular ou (c) astragalectomia com fusão semelhante. A astragalectomia por si só e imprópria de vêz que a articulação do tornozelonão tem propriocepçao. Amputação é medida de última instancia em casos de grande destruição das articulações componentes do astrágalo. Um comentario é feito sobre a fratura de avulsão espontánea do calcaneo, complicacão tabé tica rara, sequela de demanda mecanica sôbre um osso ja insuficiente por osteite sifilitica.

RÉSUMÉ

L'auteur attribue la maladie de Charcot localisée à la cheville à une destruction neuropathique à point de départ au niveau de la tête de l'astragale. Practiquèe à un stade précoce la fusion de l'arcade plantaire donne de bons résultats. Dans les cas plus avancés nous disposons de trois possibilités: a) arthrodése pan-astragaloidienne; b) fusion du pied, combinée à une fusion extra-articulaire; c) astragalectomie aves fusion analogue. L'astragalectomie seule est incorrecte, l'articulation de la cheville n'étant pas proprioceptive. L'amputation n'est indiquée que dans les cas de destruction importante.

L'auteur commente la fracture spontanée par avulsion du talon (tabès), séquelle d'un mécanique à un os affaibli (ostéite syphilitique).

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The real problem that confronts us, and it is a great problem, is how to adjust religion to science, faith to knowledge, ideality to reality, for adjustment in the reverse direction will never happen. Facts cannot be eliminated by ideals and it is too late in the history of the world to attempt to refute the findings of science by sentimental objections or supposed theological difficulties. If science makes mistakes, science must furnish the cure; it can never be done by church councils, state legislatures, nor even by popular vote.

The only possible remedy for the present deplorable condition is not less but more and better science and education; science that recognizes that the search for truth is not the whole of life, that both scientific reality and religious ideality are necessary to normal, happy, useful living. We must keep our feet on the ground of fact and science, but lift our heads into the atmosphere of ideals. "To the solid ground of nature trusts the mind that builds for aye." Education from the earliest years must teach love rather than hate, human brotherhood rather than war, service rather than selfishness; it must develop good habits of body and mind; it must instill reverence, not only for truth but also for beauty and righteousness.

—*Conklin*

Salmonella Osteomyelitis

Report of Three Cases, One With Fatal Outcome and Autopsy

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AND

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BONE and joint infections due to food-poisoning Salmonellae are more uncommon than typhoid and paratyphoid infections. Guerra[1] in 1939 reviewed the literature and collected 15 authentic cases of Salmonella bone or joint infection reported between 1926 and 1938. Only 2 cases involving the lumbar portion of the spine have been recorded. Miller[2] (1954) described a case with a fatal outcome, similar to the one to be described here, in which acute pyogenic osteomyelitis of the second and third lumbar vertebrae, psoas abscess and aneurysm of the adjacent abdominal aorta were present. The organism in this instance was S. Dublin. Ralston[3] (1955) reviewed the literature and reported a case of osteomyelitis of the lumbar portion of the spine due to Salmonella choleraesuis, and stated that this was the first recorded case of osteomyelitis of the spine due to this organism. Osteomyelitis due to S. choleraesuis, as well as to other members of the Salmonella group, has been recorded. Harvey[4] reported the cases of 71 patients with infection due to S. choleraesuis. Of this number, 15 had involvement of the bones and joints severe enough to demand clinical attention. In all of the fatal cases in which death occurred, multiple involvement of the skeletal system was recorded. Goulder and his associates[5] reported that the skeletal system is involved in 20 per cent of the cases of S. choleraesuis infection in man.

The most frequent sites of skeletal involvement seem to be the proximal end of the humerus, the distal end of the femur and the distal end of the tibia.

Three cases of proved Salmonella osteomyelitis are presented, the patients ranging in age from 12 months to 63 years.

Submitted for publication Jan. 23, 1957.

REPORT OF CASES

CASE 1 (Miriam Hospital).—F. R., a 63-year-old white man, was seen on June 14, 1954. While tightening a bolt at his regular job as a dye-setter, he lost his balance, fell backward and struck his back against a machine. He complained of localized pain in the back and was subsequently examined by his family physician (Dr. R. Rego), who referred him for orthopedic consultation. The initial examination revealed the patient to be in obvious distress, unable to stand completely erect. There was a list of the spine to the left, with complete loss of the usual lumbar lordosis. Motions of the spine were restricted in all planes. There was definite tenderness over the lumbar portion of the spine.

Roentgenograms of the lumbosacral portion of the spine, taken on June 14 (Fig. 1, *A* and *B*), revealed no evidence of recent or old bony injury. Pronounced spondylolisthesis was noted. Calcification of the abdominal aorta was observed; the aorta lay close to the spine, apparently in the normal relation. The patient was advised to obtain a back support, was given sedatives and advised to rest and apply local heat to the back. He appeared to improve; there was lessening of the pain in his back and increased motion of the spine. In September he stated that he had had severe chills, fever and increased

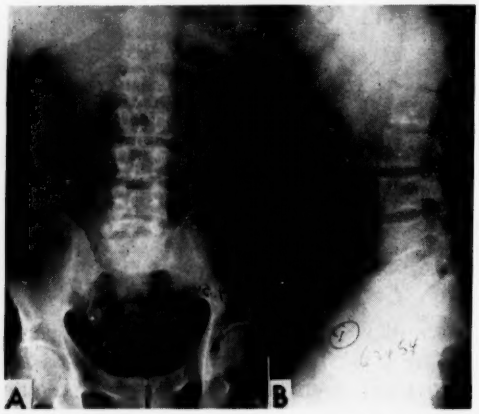

Fig. 1 (*Case 1*).—Anteroposterior (*A*) and lateral (*B*) roentgenograms of lumbosacral portion of spine taken June 14, 1945. There was no evidence of recent bony injury. Spondylolisthesis was observed. Note calcification of aorta in normal relation to lumbar portion of spine.

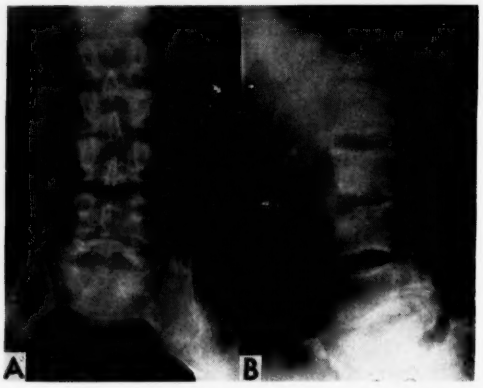

Fig. 2 (*Case 1*).—Anteroposterior (*A*) and lateral (*B*) roentgenograms of lumbar portion of spine, taken Nov. 29, 1954, showing destructive process involving bodies of second and third lumbar vertebrae, narrowing of intervertebral joint space and bony bridging between second and third lumbar vertebrae on left side. Calcified aorta has been displaced anteriorly.

pain in the back. He remained in bed for two weeks. When seen subsequently he appeared cachectic, and admission to the hospital was advised. Roentgenograms taken on admission (October 11), revealed a destructive process involving adjacent segments of the second and third lumbar vertebrae, extending to the midportion of the anterior surface of each. There was a definite decrease in the intervertebral space. Thoracic films revealed no abnormality. Laboratory studies (blood culture, stool cultures and agglutination test) revealed S. choleraesius infection. The patient was given chloromycetin (1 Gm. daily in divided doses) and made a favorable response. After the eighth day of therapy the temperature was normal and remained so until discharge, thirty days after admission. At this time there was complete absence of tenderness in the back, and motion of the spine was markedly improved. He remained at home in bed, but after two weeks the symptoms recurred, with a high temperature (103 F.) and increased pain in the back with radiation into the right groin. Chloromycetin therapy was again started, but there was no beneficial response. The patient was readmitted (Fig. 2, *A* and *B*), and the dose of chloromycetin was increased, with-

Fig. 3 (*Case 1*).—Specimen of lumbar portion of spine and abscess cavity (sagittal section). Forceps enters abscess cavity from left into saccular aneurysm on right. Extensive retroperitoneal hemorrhage.

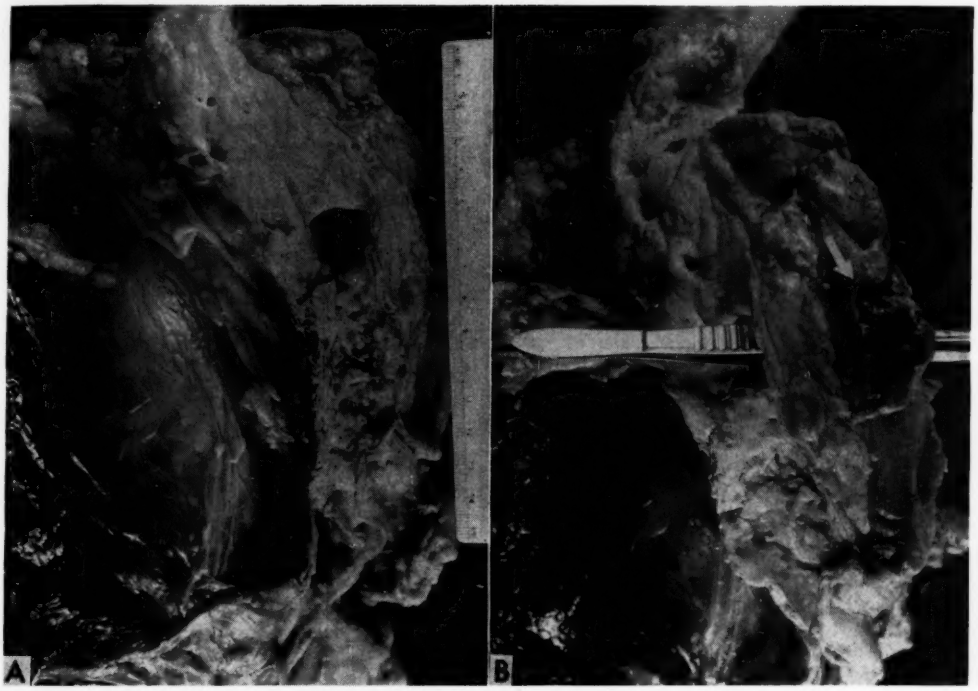

Fig. 4 (*Case 1*).—Section of aorta and retroperitoneal hemorrhage. Aorta has been incised along left margin posteriorly and reflected to right, revealing mouth of large saccular aneurysm. Psoas displaced to left by large retroperitoneal mass.

out effect on the general condition or the temperature. Operation was advised for drainage of the psoas abscess and local instillation of the antibiotic. This was attempted through a right paravertebral incision. The abscess was entered, and much detritus and granulation tissue were encountered. Cultures of this area revealed S. choleraesuis. As the cavity was enlarged a tremendous amount of arterial bleeding was encountered, which was controlled by packing, and the wound was closed with an urethral catheter *in situ*. The postoperative course was stormy. Chloromycetin was instilled locally every day through the catheter into the abscess cavity. The patient died suddenly on the tenth postoperative day.

Laboratory Data.—On admission (October 18) the erythrocyte count was 3,600,000 per cubic millimeter of blood, with 71 per cent hemoglobin; the leukocyte count was 5,200, with 64 per cent polymorphonuclears, 32 per cent lymphocytes, 2 per cent eosinophils and 2 per cent juvenile cells. On November 9 the value for hemoglobin was 77 per cent and the erythrocyte count 3,720,000 per cubic millimeter of blood. The leukocyte count was 3,400, with 58 per cent polymorphonuclears, 41 per cent lymphocytes, and 1 per cent eosinophils. On the second admission (December 12) the erythrocyte count was 3,100,000, with 65 per cent hemoglobin, and the leukocyte count was 5,300, with 83 per cent polymorphonuclears, 16 per cent lymphocytes and 1 per cent juvenile cells. Postoperatively there was a marked drop in hemoglobin and the erythrocyte count, which responded to transfusion. The white blood cell count varied between 3,000 and 4,000 per cubic millimeter, with a marked increase in polymorphonuclears. The agglutination tests for paratyphoid were positive, ranging as high as 1:5,120. Two blood cultures were positive for S. choleraesuis and became sterile after the first course of chloromycetin. Subsequently, and including the second admission,

the blood cultures were sterile. One stool culture was positive and several were negative before and after this culture was obtained. The cultures were identified by the National Salmonella Institute as S. choleraesuis, variety Kunzendorff.

Roentgenograms taken on November 5 and 29 and December 15 revealed no significant alteration in the joint space. There appeared to be gradual increase in density of the bone surrounding the area of destruction in the second and third lumbar vertebrae, which was interpreted as a repair process.

Autopsy revealed a ruptured sclerotic and mycotic saccular aortic aneurysm. Degeneration and abscess formation of the disc were noted between the second and third lumbar vertebrae (Figs. 3 and 4). S. choleraesuis was cultured from this area. The saccular aneurysm had eroded through the third lumbar vertebra and had ruptured. Extensive hemorrhage was present retroperitoneally, mainly along the right psoas muscle. Microscopic examination revealed the eroded wall of the aneurysm and the adjacent vertebrae showing partly necrotic fragments of the aortic wall and underlying bony tissue. The latter showed evidence of resorption and chronic inflammation. The inflammatory infiltrate was composed largely of plasma cells, lymphocytes and macrophages. The fusiform sac along the left lateral aspect of the second and third lumbar vertebrae had a fibrous lining, which showed a dense infiltration of lymphocytes, plasma cells and hemosiderin-laden macrophages. A few islands of residual degenerating striated muscle were also present. The hemorrhagic muscle bordering on the large peritoneal hematoma showed evidence of necrosis, dense polymorphonuclear infiltration and hemorrhage.

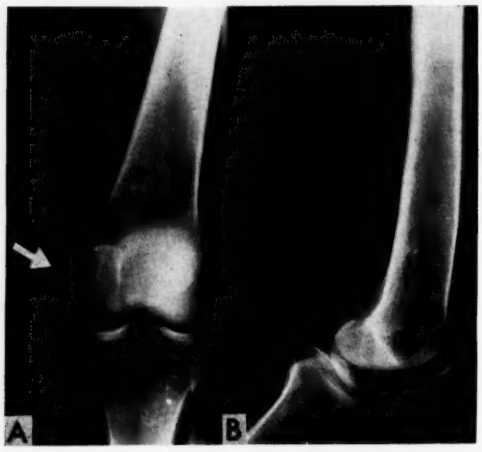

Fig. 5 (*Case 2*).—Roentgenograms of left knee taken April 29, 1953, revealing area of decreased density in medial femoral condyle. Note thinning of cortex on medial side.

CASE 2 (Miriam Hospital).—A 27-year-old white woman was seen at the office on April 29, 1953, with a history of pain in the left knee of three weeks' duration. She recalled no antecedent trauma or infection. The pain had been severe, was not controlled by salicylates and was localized at the medial aspect of the lower end of the femur. There were no episodes of locking or instability. The patient had had psoriasis approximately one year earlier and had recently noted several patches in the region of the left knee.

Examination revealed no gross swelling, redness or discoloration of the left knee, but there were several small patches of psoriatic lesions in this region. Extreme flexion of the left knee was painful. Palpation revealed exquisite tenderness over the left medial femoral condyle. No other tender areas were noted. There was no inguinal lymphadenopathy.

Roentgenograms of the left knee (Fig. 5), revealed an area of decreased density in the left medial femoral condyle. There appeared to be thinning of the cortex. The area measured approximately 1½ inches (3.7 cm.) in diameter. The joint surfaces were smooth and regular. No soft tissue calcification was noted.

The patient was admitted to the Miriam Hospital, and on May 4, through a 3 inch (7.5 cm.) incision over the medial femoral condyle, the site of the lesion was exposed. The periosteum stripped easily. Under roentgen control, the lesion was identified. A window was cut in the cortex and an abscess cavity, measuring 1 by 2 inches (2.5 by 5 cm.) was revealed. The cavity contained thick white material mixed with red granulomatous material. Thorough currettage was performed. The wall of the cavity was hard. Closure was effected in layers. Prior to closure, penicillin (1,000,000 units) was instilled into the wound and the bone cavity. The postoperative course was uneventful, and the patient was discharged on May 10. The sutures were removed on the ninth post-

operative day. The wound appeared to be well healed, but a slight increase of warmth in the left knee was observed. There was tenderness over the left inguinal lymph nodes. Chloromycetin was continued. On May 16, drainage of yellowish-brown watery matter from the wounds was observed. The patient was then given terramycin. The wound continued to drain in small amounts. Cultures of the wound July 16 revealed no evidence of Salmonella; nonhemolytic staphylococci and Proteus vulgaris were identified. A subsequent culture on August 7 revealed Salmonella. Drainage ceased on September 3, and there has been no recurrence of symptoms since. The patient was seen on Nov. 16, 1954, and had no complaints referable to the knee. Motions were free and painless. Roentgenograms (Fig. 6) revealed the operative site to be clearly visualized and the surrounding bone normal. No evidence of inflammatory reaction was noted.

Fig. 6 (*Case 2*).—Roentgenograms of left knee taken Nov. 16, 1954. Operative site is clear. Surrounding bone of medial femoral condyle shows no abnormalities.

Laboratory Data.—On admission to the hospital (October 18) the erythrocyte count was 4,160,000 per cubic millimeter of blood, with 71 per cent hemoglobin; the leukocyte count was 8,600 per cubic millimeter, with 67 per cent polymorphonuclears, 29 per cent lymphocytes, 2 per cent eosinophils, 1 per cent polymorphonuclear band cells and 1 per cent juvenile cells. Urinalysis gave essentially negative results. The blood chemical values (sugar, urea nitrogen, total protein, albumin, globulin, calcium, phosphorus and alkaline phosphatase) were within normal limits. The organism revealed by culture of the bone abscess was identified by the National Salmonella Institute as S. oranienburg. One stool culture was negative.

CASE 3 (Rhode Island Hospital)*.—W. J. L., a 12-month-old Negro boy, was admitted to the hospital on Oct. 10, 1955. The chief complaint was of fever and pain in the right leg. The patient's mother stated that he had been in his usual state of health until one day prior to admission, when she noted that his skin was hot and that he appeared to favor the right leg. The fever responded to acetylsalicylic acid, but the following day the mother again noted that the child had fever and seemed to have a painful right leg. There was no history of vomiting or diarrhea and no family history of tuberculosis. On admission the temperature was 103.6 F., the pulse rate 120 and the respiratory rate 130. The child was well nourished and well developed. He cried when the right leg was manipulated. The skin was hot and dry, and there was no rash. The ears, nose and throat were clear. The lungs were clear. Examination of the heart revealed no abnormality. Abdominal examination also gave negative results. There was pain on motion of the right hip joint with limitation of motion of the hip in all directions, as well as definite splinting when walking or weight-bearing was attempted. The left hip was normal.

Laboratory Data.—On admission, the value for hemoglobin was 8.6 Gm. There were 12,300 leukocytes per cubic millimeter of blood, with 40 per cent polymorphonuclears, 52 per cent lymphocytes and 8 per cent monocytes. Urinalysis gave negative results. The value for fasting blood sugar was 80 mg., and that for urea nitrogen 14 mg., per hundred cubic centimeters. A Hinton test gave negative results. Roentgenograms of the chest revealed no abnormality.

Roentgenograms of the pelvis and hips revealed no bony pathologic change (Fig. 7*A*). Lumbar puncture showed the protein content of the spinal fluid to be 15 mg. and the dextrose content 85 mm. per hundred cubic centimeters with no cells. Nose and throat cul-

*Service of Dr. Kenneth G. Burton, R. I. Hospital.

ture revealed strepviridans, neisseria, diphtheroids and Aerobacter aerogenes. The spinal fluid was negative for acid-fast organisms. Culture of the spinal fluid gave negative results. Agglutination tests showed paratyphoid H 1-40; typhoid 0 1-80. Three blood cultures revealed S. typhosa. Stool cultures repeatedly gave negative results for pathogenic organisms.

Clinical Course.—The child was placed in barrier and continued to run a high temperature (102 to 104 F.) for the first week. During the second week and the course of chloromycetin the temperature came down and remained in the range of 99 to 101 F. Because of a decreased leukocyte count during the third week chloromycetin was discontinued and terramycin administered, after which the temperature again rose to levels of 102 and 103 F. and, on one occasion, to 105 F. On the twenty-first day after admission all antibiotics were discontinued for the next four days. The temperature continued at 103 to 104 F. but gradually subsided. By the twenty-ninth day after admission the child was afebrile and remained so until his discharge, except for two transient elevations of the temperature to 101 F. Repeated blood cultures were negative. Repeated roentgenograms of the chest, pelvis and both hips revealed no abnormality and no signs of productive or destructive bone disease.

On November 26, 1955, the child was noted to have chickenpox and was discharged home to return to the orthopedic clinic. Roentgenograms (Fig. 7*B*) taken on December 24 revealed a fairly large area of decreased density involving the upper portion of the right femur just distal to the epiphyseal plate. A plaster spica was applied and was removed for further roentgen examination on Feb. 23, 1956 (Fig. 7*C*). There was definite improvement in the roentgen appearance, with evidence of partial healing of the area of decreased density. On July 10 roentgenograms revealed complete absence of the rarefied area (Fig. 7*D*) with evidence of sclerosis indicative of advanced healing. Some broadening and shortening of the right femoral neck was noted.

COMMENT

The genus Salmonella comprises gram-negative and non-sporeforming bacilli which are, as a rule, motile by means of peritrichous flagellae. They have one common quality: all the known species are pathogenic for man, animals or both.

Salmonella infection in man is usually one of three types: (1) gastroenteritis, varying from a mild attack to a severe type ending in death; (2) Salmonella fever, a typhoid-like febrile illness, or (3) Typhoid septicemia, the symptoms and signs in individual cases depending upon the location of any abscess.

The organism usually responsible for the third type is one belonging to Salmonella Group C, commonly S. choleraesuis. Intestinal symptoms and lesions are usually absent, and the organism is rarely recovered from the stool.

Diagnosis and identification depend upon isolating the organism from the blood or from the focus of infection.

In Case 1 the organism was isolated from the blood, the stool, the localized area of abscess at the time of operation and at autopsy. In Cases 2 and 3 cultures of the stool were negative. In Case 2 the organism was isolated from the area of abscess, and in Case 3 repeated cultures of the blood were positive.

Edwards and his associates[6] have reported 3,016 cultures of Salmonella organisms recovered from human sources. Of these, 151 (5 per cent) showed S. choleraesuis. Of the total group from human sources, 184 were detected in blood cultures, and 84 of these (45.6 per cent) were S. choleraesuis. There were 56 cases of fatal outcome, and S. choleraesuis was the organism in 24 (42.8 per cent). Saphra[7] reported a mortality rate of 5.3 per cent from the entire Salmonella group, but for S. choleraesuis the rate was 21.3 per cent. Figures approximating these have been reported by others, but it is interesting that, of 149 cultures of this organism of known origin, not one was isolated from a normal human carrier.

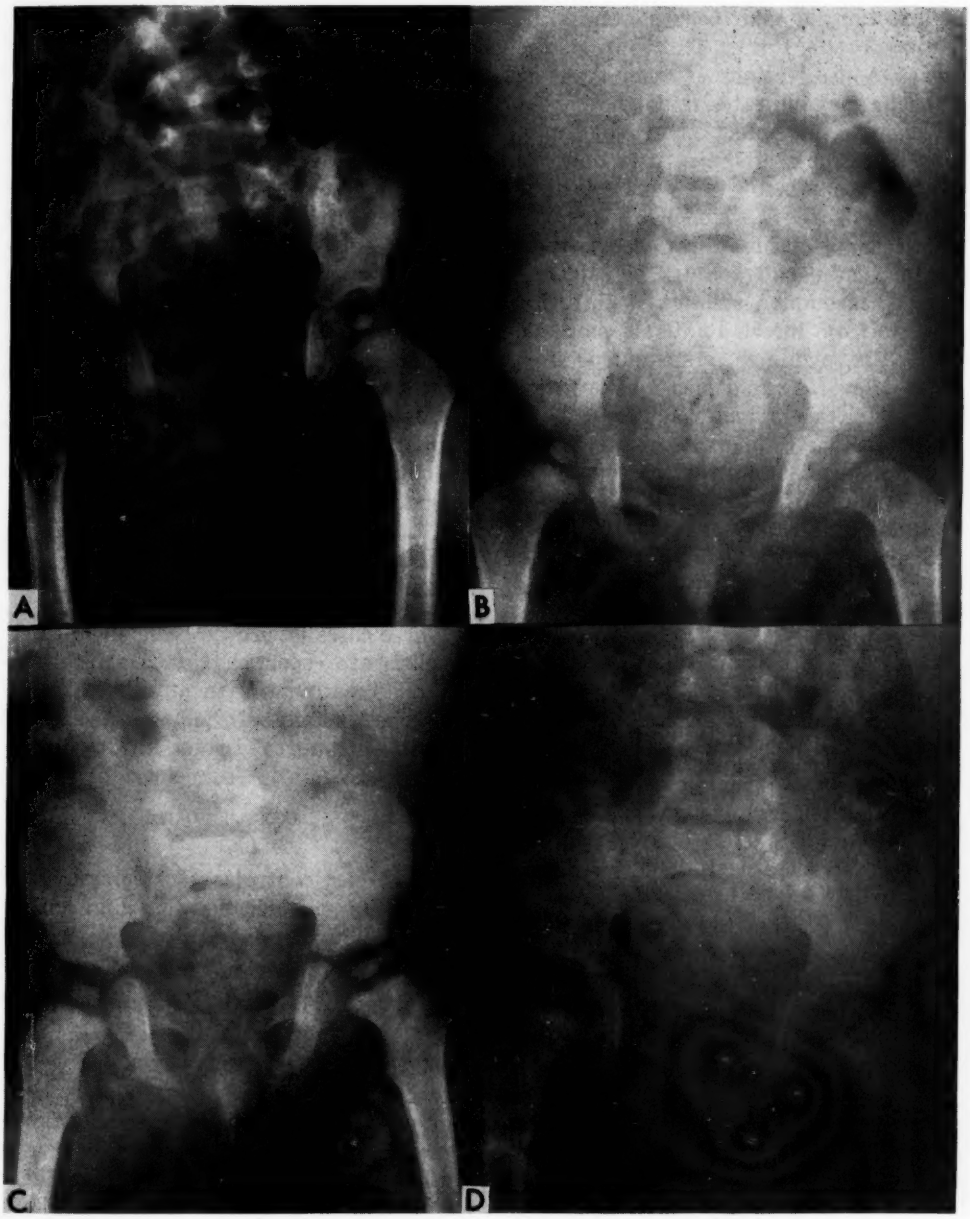

Fig. 7 (*Case 3*).—*A*, roentgenogram of pelvis taken Oct. 29, 1955. No pathologic changes of bone are revealed. *B*, film taken Dec. 24, 1955, showing large area of decreased density in upper portion of right femur, distal to epiphysis. *C*, film taken Feb. 23, 1956, after removal of hip spica. Definite improvement is observable, with diminution of area of decreased density. *D*, film taken June 5, 1956. Note complete absence of rarefied area with evidence of sclerosis, indicative of good healing.

These figures indicate that S. choleraesuis infection in man occurs almost always in spofadic form. This is in strong contrast to observations of other types of Salmonella and indicates that the sources of S. choleraesuis infection in many may not be the same as those of infection due to other members of the genus. Other organisms of the Salmonella genus are responsible for the more common type of enteritis that usually occurs in epidemic form. The common sources of infection are meats from infected animals, sausage, cream puffs, puddings, milk and polluted water. Several epidemics have been traced to duck eggs, and isolation of many members of the Salmonella genus in dry egg powder has suggested that this is the possible source of infection.

Contaminated foods are not altered in appearance, taste or odor, and the presence of such contamination can be determined only by bacterial examination. A symptom-free carrier is a source of potential danger.

The effect of chloromycetin was not striking in any of the cases. Initially there was a good response in Case 1, but subsequently the drug failed to control the infection. Terramycin was also used in Cases 2 and 3.

In Case 1 the localization of the disease to the lumbar portion of the spine could be explained as due to metastasis or to hematogenous osteomyelitis. Weaver and Sherwood[8] stated that hematogenous osteomyelitis due to S. suipestifer apparently occurs only in infants. (This probably applies to Case 3.) A more likely explanation lies in the association of the lesion with aneurysm of the abdominal aorta. (A. A. Miller[2] stated that the repeated and continued erosion of the lumbar bodies by a large aneurysm would provide a locus minoris resistentiae, with local infection by the organism and a projection of the acute osteomyelitis.) Most agree that, in the production of typhoid, paratyphoid osteitis and osteomyelitis, injury is an important factor.

One must consider also that the psoas abscess could have caused the aneurysm. Microscopic examination of the aneurysm revealed vascular obstruction in its wall. Serial roentgenograms further substantiate this theory. Films taken three weeks after the injury revealed no evidence of involvement of the lumbar portion of the spine. Subsequent roentgenograms revealed destruction of the second and third lumbar vertebrae, and one might speculate that injury to the spine caused a local lowering of resistance with the onset of hematogenous osteomyelitis, abscess formation and erosion of the wall of the abdominal aorta (due to pressure of the abscess), with an eventually fatal outcome.

CONCLUSION

Three cases of proved Salmonella osteomyelitis are reported, 1 with a fatal outcome. Treatment with antibiotics failed in 1 case, undoubtedly helped in the second and was of unproved value in the third.

CONCLUSIONS

Trois cas contrôlés d'ostéomyélite de Salmonella sont décrits, dont l'un mortel. Les antibiotiques ont échoué dans un cas, ont été très utiles dans le deuxième, et d'une valeur non ètablie dans le troisième.

SCHLUSSFOLGERUNGEN

Es wird über drei Fälle von nachgewiesener durch Salmonellaerreger hervorgerufene Osteomyelitis berichtet, von denen einer tödlich ausging. Die Behandlung mit Antibiotika versagte in einem Falle, war zweifellos wirksam in zweiten Fall und von nicht nachweisbarer Bedeutung im dritten Fall.

CONCLUSAO

3 casos provados de osteomielite por Salmonela são apresentados, dos quais um foi fatal. O tratamento com antibioticos falhou em um caso, sem dúvid auxiliou no segundo caso e de valor não comprovado no terceiro.

CONCLUSION

Se presentan 3 casos de osteomielitis originados incuestionablemente por salmonellas, uno de ellos seguido de éxitus. El tratamiento con antibióticos falló en un caso, ayudó indiscutiblemente en el segundo y fue de discutible valor en el tercero.

CONCLUSIONI

Vengono riferiti tre casi certi osteomielite da Salmonella uno dei quali infausto. In uno di essi la cura con antibiotici fu inefficace, mentre nel secondo certamente giovò e nel terzo fu di incomparabile giovamento.

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The specialist may thrive on knowledge and skill; without wisdom the consultant is useless. The specialist provides the detailed information. He is the permanent expert, the head of a civil service department, while the consultant is the cabinet minister who, guided and helped by the experts, must decide policy.

—*Ogilvie*

Technic and Indications for Arthroplasty of the Hip with Acrylic Prostheses

Review of Final Results

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UTRECHT, THE NETHERLANDS

ARTHROPLASTY by means of an acrylic prosthesis as suggested by the brothers Judet was introduced at a time when the cuparthroplasty of Smith-Petersen had begun to give disappointing results. The initially favorable results of the Judet method drove some enthusiasts so far that they performed this operation on every pseudarthrosis of the femoral neck and even on fresh fractures.

Within a short time, extensive material could be collected from the world literature. It soon enabled the orthopedic surgeons to base their judgment of arthroplasty on a great number of cases. On the strength of theoretical and biologic considerations, some of them, being critically-minded, had already warned against the use of this procedure. Even the preliminary results (by which I mean the results up to three years after the operation) showed that the greatest reserve is necessary in considering the Judet arthroplasties, and that the indications should be extremely sharp before this operation is employed.

As early as 1951 and 1953 I tried to arrive at an accurate formulation of indications for the Judet arthroplasty based on my personal material, with the intention of obtaining a considerable decrease in the number of poor results. My follow-up in 1956 of 225 arthroplasties showed that the Judet arthroplasty, restricted indications, still deserves to be maintained, as this most objective investigation yielded 52.7 per cent of good results.

Wishing to distil the causes of failure from this follow-up in order to work out an accurate scheme of indications, I must admit that the investigation let me down to a great extent. Although there were some striking exceptions, almost no regularity could be observed in the factors exerting a favorable or unfavorable influence on the arthroplasty. In the period from 1947 to 1956 I performed 225 arthroplasties of the hip. The follow-up, conducted to determine the value of the Judet arthroplasty, was carried out in June and July, 1956. Various methods are available in the literature, among them those of Gade, Judet and Shepherd and the much-used one of Merle d'Aubigné. It cannot be denied that all of them, mine included, showed defects. Still I preferred my method (Verhandlungen der Deutschen Orthopädischen Gesellschaft, 1956), as it better accentuates the factors that are important in evaluating the arthroplasty.

I used two schemes, one for the clinical follow-up and the other for roentgen examination. The results were expressed in the figures. In the clinical follow-up the following factors were successively calculated: (1) the position of the hip-joint; (2) the function of the hip-joint; (3) the

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static sufficiency of the hip-joint; (4) the muscular strength, and (5) the pain.

The roentgen examination was subdivided as follows: (1) changes in the canal of the stem; (2) changes in the supporting plane of the prosthesis; (3) changes in the acetabulum; (4) changes in the periarticular tissue, in particular ossifications, and (5) fractures and other defects of the prosthesis.

These investigations afford a good impression (Table 2) of the condition of the hip-joint both clinically and roentgenologically, and the results are comparable.

In performing the Judet arthroplasty I prefer the Smith-Petersen incision, without detachment of the rectus femoris muscle. It gives an excellent approach to the joint, particularly when one is dealing with subluxation of the hip with anteversion of the head. It was Max Lange who rightly remarked that this incision should always be used in such cases.

The Smith-Petersen incision was employed in 187 cases. In 40 cases the Gibson approach or its modifications were used. In some cases the classic approach of Lexer Murphey was followed, but, like the Gibson incision, it carries the disadvantage that the major trochanter must be detached. It is possible to avoid this by using Merle d'Aubigné's or Chapchal's method. The lateral or the posterior approach still results in poor exposure. The molding of the acetabulum from this approach is difficult.

In the beginning I always completely resected the head, but I soon arrived at the conclusion that this should not be done, and that only the upper calotte should be removed, so that the femoral neck is extended. It counteracts the postoperative insufficiency of the gluteal muscles.

Only in the beginning was the prosthesis placed axially in the femoral neck, as described by the brothers Judet. With this technic, however, the lower part of the collum soon atrophies, owing to the strong pressure of the prosthesis, the canal becomes wider and the pin assumes a practically horizontal position, so that the acetabulum rests on the upper angle of the collum. For these reasons I inserted the stem of the prosthesis steeply, at any rate in the direction of the medial corticalis of the femoral neck, so that the prosthesis was placed in valgus. Never must the stem of the prosthesis be fixed in the lateral corticalis of the femur; it has to point free into the spongiosa of the trochanter. This arrangement guarantees a better division of the pressure and causes considerably fewer changes in the canal. These technical demands were published and recommended at the Orthopedic Congress of Heidelberg in 1951, and the method was adopted by many colleagues. It is interesting to mention that, of the prostheses inserted in varus, only

TABLE 1.—*Indications*

Arthrosis Deformans		91
Luxatio coxae congenita		58
Coxa vara		6
Otto-chrobak		8
Trauma coxae		29
Luxatio coxae et necrosis capitis	10	
Pseudarthrosis colli femoris	19	
Ankylosis coxae		16
Coxitis nonspecifica	3	
Morbus Bechterew	5	
Polyarthritis	6	
Smith-Petersen		13
Judet		6
68 ♂ + 157 ♀	=	225

TABLE 2.—*Follow-up, 1956*

	Clinical Results	*Roentgen Results*
Good	52.7	38.2
Fair	36	54.2
Poor	11.3	7.6

13 per cent gave favorable and 87 per cent poor results; of those inserted in the neutral position 25 per cent gave good and 75 per cent poor results, but of the prostheses inserted in valgus 66 per cent gave favorable and only 34 per cent poor results.

The capsule was completely resected apart from the capsula reflexa at the femoral neck, as it contains the blood supply of the femoral neck. This gave good results. Periarticular calcifications were then encountered in only 17 per cent of cases and gave rise to functional troubles in less than 7 per cent. The removal of the cartilage from the acetabulum, as recommended by Smith-Petersen, was not employed, as I had not observed very good results from it and soon realized the advantages of preservation of the cartilage.

If the acetabulum was insufficiently developed a shelf operation subsequent to the arthroplasty was highly satisfactory, while deepening of the acetabulum gave poor results.

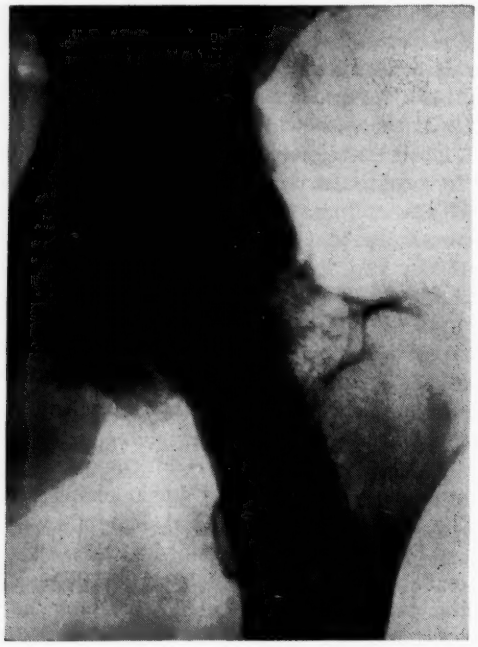

Fig. 1.—Example of a good arthroplasty.

Apart from that, 50 per cent of the arthroplasties showed necrosis of the acetabulum. I have already reported, in extensive articles, on the indications to arthroplasty. Here I need not enlarge on the necessity of a general limitation of these indications. The patients should be rigidly selected, not only from the physical but from the psychic standpoint. Arthroplasty should not be performed (1) on a young patient with a still growing skeleton; (2) in the presence of atrophy or dystrophy of the upper end of the femur; (3) on a patient with poorly developed or atrophic muscles; (4) on a patient in poor general condition, physically or mentally, and (5) in the presence of inflammatory conditions of the hip, including rheumatic arthritis and Pierre Marie's disease.

In an article in this *Journal* (1951) I dealt extensively with the question of atrophic cysts of the hip in connection with arthroplasty. For this atrophic form of arthrosis deformans arthrodeses may deserve recommendation. For osteoarthritis, arthroplasty according to Judet is indicated only if the disease has not given rise to serious atrophy of the upper end of the femur, as otherwise the prosthesis eventually assumes a varus position under the influence of weight bearing. When in such a case the prosthesis is in a valgus position, it can give way so deeply, owing to necrosis of the supporting plane, that the musculature becomes insufficient. Great accuracy, therefore, is required as regards the indications.

I observe great caution with arthroplasty in the treatment of malformations. In the first place, if there is congenital luxation of the hip with the well-known anteversion of the head, it is not easy to fix the prosthesis, unless a prosthesis of the Gosset type is used.

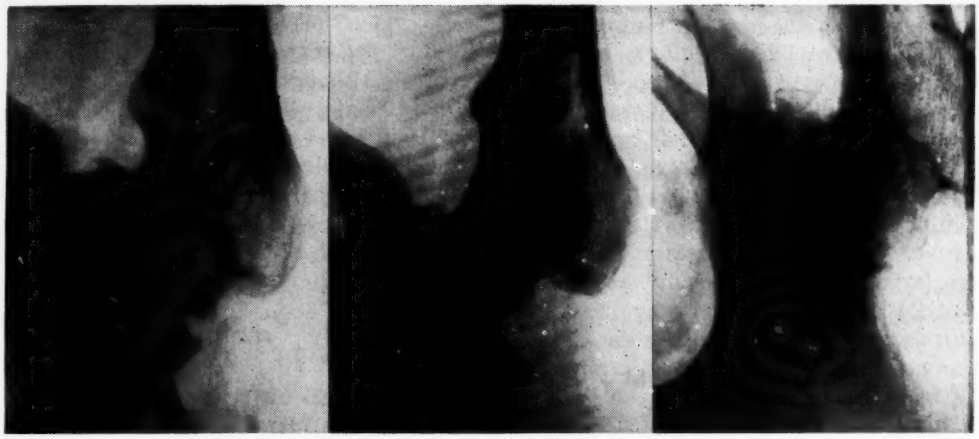

Fig. 2.—Judet arthroplasty for osteoarthritis; preoperative roentgenogram, well-fitting prosthesis half a year after the operation, and necrosis of the femoral neck and acetabulum three and one-half years after the operation.

There are two absolute osseous contraindications, namely, dystrophy of the upper end of the femur as observed after inflammations and in cases of arthrosis deformans and of primary congenital inferiority of the osseous tissue and the immature bone. In young patients the indications for correction of epiphysiolysis or coxa vara are much exaggerated and extremely bad. In general, arthroplasty should not be performed on young patients, as young bone does not tolerate substitution of the prosthesis and the implantation of foreign material and rapidly atrophies under its influence.

TABLE 3.—*Diagnosis*

Diagnosis	% of 225 cases	*Good*		*Fair*		*Poor*	
		% *Cl. Res.*	% *Rad. Res.*	% *Cl. Res.*	% *Rad. Res.*	% *Cl. Res.*	% *Rad. Res.*
Arthrosis deformans	43	38.5	28	38.5	59	23	13
Dysplasia coxae	15	33	50	59	42	8	8
Trauma coxae	15	85	62	15	38		
Polyarthritis	8	17	50	50	17	33	33

The indication of choice for arthroplasty is a post-traumatic lesion of the femoral head. Arthroplasty is indeed to be preferred when the head has become necrotic or is fractured as a result of the trauma. In other cases, in which the head has retained its vitality, Brackett's procedure is still preferable to the prosthetic method, as any technic in which living tissue is preserved is always better than one which introduces foreign material.

It is known that various metacrylates are indeed not inert with respect to living tissue and that they may provoke acrylosis, just as certain kinds of metal may produce metallosis. It is also known that various prostheses are not strong enough, or readily wear away at the places subject to the greatest stress, or show corrosion under the influence of the tissue fluid; the last-mentioned, particularly, happens in the grooves of the pin. The corrosion gives rise to early fracture of the material. The plexiglas used for my prostheses (trademark Pekaro) is completely unaffected by living tissue, and it is so strong that in this series practically

no fractures of the pin occurred, and only exceptional wearing away of the head was observed. As regards fracture of the pin my figures are decidedly favorable, as I meet the mechanical demands by implanting the prosthesis steeply. I am extremely cautious with metal, as it has been shown that no metal is inert with respect to living tissue. It may be that certain alloys soon depolarize, although it is not improbable that these metals repolarize again on certain oscillations of the pH in the tissues. One must most certainly bear in mind the differences in potential of the metal, the prostheses being usually situated in different milieus.

Before proceeding to a summary of the results of this follow-up, I wish to point out that my general policy as regards the indications for arthroplasty has proved correct. It is evident that the arthroplasties performed on patients with rheumatoid arthritis and after Bechterew's disease, in which stiffening is usually rapid, and after tuberculous coxitis gave bad results. The tables show the results for the various indications. The advantages of arthroplasty for post-traumatic complications are outstanding. Arthrodesis is to be recommended for patients whose work involves heavy demands on the hip joint.

SUMMARY

The indications for arthroplasty must be considerably restricted. This attitude should certainly be adopted toward malformations of the hip joint, but also toward osteoarthritis. The border between indications and contraindications is not yet quite clear, but it can be contended that the indications hitherto considered ample cannot be maintained. This does not imply that arthroplasty should be completely abandoned. Our follow-up of a group of patients so treated under rather restricted indications revealed good results in 52.7 per cent of cases, fair results in 36.0 per cent, poor results in 11.3 per cent. Although many follow-ups on arthroplasties have been published, the last word has not yet been said on this subject. It is known, however, that arthroplasty can be maintained as an asset in orthopedic surgery if a wise limitation is observed with regard to the indications.

RÉSUMÉ

Les indications de l'arthroplastie doivent être considérablement restreintes. Ce principe devraitrêtre adoptésaussi bien à l'égard des malformations de l'articulation de la hanche qu'à l'égard de l'ostéoarthrite. La limite entre les indications et les contre-indications n'est pas encore bien définie, mais il est évident que les indications étuendues encore en vogue aujourd'hui ne peuvent être maintenues, ce qui ne signifie pas que l'arthroplastie doive être abandonnée. Les observations portant sur un groupe de malades traités dans le cadre d'indications assez limitées ont montré 52.7% de bons résultats, 36% de résultats passables, et 11.3% de résultats médiocres. De nombreux cas d'arthroplasties ont été publiés à ce jour mais tout n'a pas encore été dit sur ce sujet. L'arthroplastie est un atout précieux en chirurgie orthopédique, mais à condition d'en limiter avec prudence les indications.

RIASSUNTO

Le indicazioni all'artroplastica devo essere notevolmente ristrette. Questo principio deve essere seguito nei confronti delle malformazioni dell'articolazione dell'anca e dell'osteoartrite. Il confine fra indicazioni e controindicazioni è lungi dall'essere stabilito, ma le indicazioni devono sicuramente essere ristrette. Questo non significa che l'artroplastica debba essere

completamente abbandonata. Un controllo eseguito su un gruppo di pazienti così trattati, con indicazioni abbastanza ristrette, dimostrò buoni risultati ned 52,7% dei casi, risultati discreti nel 36% e cattivi nell'11,3%. L'ultima parola su questo argomento non è ancora stata detta. E' certo, però, che l'artroplastica deve essere conservata fra i mezzi ortopedici a patto che le sue indicazioni vengano limitate con prudenza.

RESUMEN

La indicaciones de la artoplastia deben ser restringidas considerablemente. Esta actidud debe ser efectivamente adoptada en el caso de las malformaciones de la cadera pero también en los casos de osteoartritis. El límite exacto entre indicaciones y contraindicaciones no está todovía claro pero puede afirmarse que la indicación para tan amplio número de casos como venía haciéndose no puede ya mantenerse. Ello no implica el que la artoplastia deba ser abandonada completamente. La investigación ulterior en unsa serie de individuos tratados así en un grupo mas bién restringido de indicaciones revela buén resultado en un 52,7 por ciento do los casos, siendo aceptable en un 36 por ciento y malo en un ll por ciento. Aunque se han publicado muchas observaciones ulteriores de artroplastias aún no se ha dicho la última palabra en esta materia. Se sabe de todas maneras que la artroplastia puede ser considerada como de un valor real en cirugía ortopédica si e observa una limitación inteligente en relación con las indicaciones.

ZUSAMMENFASSUNG

Die Indikationen zur Gelenkplastik müssen erheblich eingeschränkt werden. Diese Forderung ist besonders gerechtfertigt in Fällen von Missbildungen des Hüftgelenks, in gewissem Umfang aber auch bei Osteoarthritis. Wenn sich auch noch keine scharfe Grenze zwischen Indikationen und Gegenindikationen ziehen lässt, so kann doch gesagt werden, dass der bisher angenommene Umfang der Indikationen nicht aufrecht erhalten werden kann. Das bedeutet nicht, dass die Gelenkplastik völlig aufgegeben werden soll. Die Nachuntersuchung einer Gruppe von Kranken, die mit Gelenkplastik unter ziemlich eingeschränkter Indikationsstellung behandelt wurden, ergab gute Resultate in 52,7 Prozent, mässige Resultate in 36,0 Prozent und schlechte Resultate in 11,3 Prozent. Trotz der zahlreichen Veröffentlichungen von Nachuntersuchungen in Fällen von Gelenkplastik ist das letzte Wort über dieses Thema noch nicht gesprochen. Wir wissen jedoch, dass die Gelenkplastik ihren Platz in der orthopädischen Chirurgie behalten kann, wenn wir uns hinsichtlich ihrer Indikation Beschränkungen auferlegen.

SUMARIO

As indicações para a artroplastia devem ser consideràvelmente restringidas. Esta atitude deve tambem ser adotada com relação as mal formações da articulação coxo- femural, porém também para as osteoartrites. Os limites entre indicações e contra indicações ainda não estão perfeitamente definidos, porém desde já as amplas indicações atuais não podem ser mantidas. Tal conceito nao implica que o método de artroplastias deve ser completamente abandonado. O estudo pos operatório de um grupo de pacientes para os quais as indicações obedeceram o plano mais restrito, revelou bons resultados em 52.7% dos casos, aceitaveis em 36.0% e resultados pobres em 11.3%.

Ainda que muitos estudos dos resultadoscom artroplastias tenham sido publicados, a última palavra no assunto não foi conseguida.

É sabido no entanto que a artroplastia pode ser mantida como metodo cirúrgico ortopédico se uma limitação inteligente for observada com relação as indicações.

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Management of Foreign Matter in the Anterior Segment of the Eye, with Particular Reference to Occupational Injury

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FOREIGN matter in the anterior segment of the eye is a major problem in industry. This paper reviews methods of diagnosis and treatment of intraocular injury affecting the lids, the cornea, the conjunctiva, the sclera and the area anterior to the ciliary body and the zonule of Zinn but including the lens.

When taking a history, the physician should ask the patient a few pointed questions that can help identify the foreign material involved and indicate the correct surgical procedure.

"What kind of material were you working with?" If possible, a sample of the material that injured the patient should be brought to the office and tested for magnetism. The radiopaqueness of glass varies with its composition. Thus, if the foreign body is suspected to be glass a sample can aid the roentgenologist in determining his technic for demonstration and localization of the foreign body in the eye.[3]

"What was your vision before this accident?" If possible, the patient should be cross-checked with a pre-employed or other periodical examination record, for when compensation is involved patients will sometimes exaggerate the loss of vision due to an accident.

"When did the accident occur?" If too much time has elapsed between the injury and removal of the foreign body, there may be a more severe reaction and a less favorable prognosis.

"What were you doing when the accident occurred?" Details of the position of chiseling, hammering or rapidly rotating machinery give an indication of the speed of impact and the character of the foreign material involved.[4]

Examination of the patient begins with the determination of his visual acuity; a record of the findings is made for each eye *both with and without glssses.* If the visual acuity is less than 20/20 or 6/6, the vision is checked with a pinhole disc. If blepharospasm is present, it is best to instill a topical anesthetic agent such as tetracaine hydrochloride (0.5 per cent) before determining the visual acuity.

Both eyes are then examined with a focused light and a magnifying loupe. Often more than one foreign body is present. A careful search is made for a perforating wound of entry, as shown by a nebulous corneal scar, a hole in the iris or a spot of subconjunctival hemorrhage. A drop of fluorescien (1 or 2 per cent) followed in a

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minute by irrigation with physiologic solution of sodium chloride or boric acid solution will stain a recent corneal injury or encircle a foreign body with a green stain, making it more visible. Any small wound in the cornea must be considered the wound of entry of a foreign body until proved otherwise.

A foreign body in the cul-de-sac of the conjunctiva may be seen by eversion of the lid. If the foreign body is not seen, and the cul-de-sac of the upper lid is not exposed by pressure on the globe through the lower lid, the folded conjunctiva is lifted for a double eversion with a Desmarres' or a Walker's lid speculum.[5]

The lacrimal puncta should be observed with a loupe and good illumination, for one may see in the orifices thereof a foreign body.

A transilluminating light placed on the lower lid while the iris is observed will clearly show a hole in the iris or iris atrophy.

After the pupil is dilated with a mydriatic, the ophthalmoscopic examination is made to find a shadow of the wound of entry as well as a foreign body and cataract.

The slit lamp is a valuable tool in management of such cases, since it helps the practitioner judge the anatomical depth to which the foreign body has penetrated, and enables him to observe the progress of removal and postoperative healing. Very small foreign bodies, especially particles of glass, can be removed under slit lamp observation, but such a procedure is awkward and tedious and is followed only in special cases.[6]

Gonioscopic study may bring out a hidden foreign body in the angle of the anterior chamber not demonstrable by any other method of examination. Any person with an unexplained iridocyclitis should have the benefit of a gonioscopic examination to rule out an angle foreign body.[7]

It is important to determine ocular tension. A soft eye suggests a perforating wound.

The Berman locator is useful in finding hidden magnetic foreign bodies in the anterior segment of the eye. It is especially useful when an experienced roentgenologist is not available. Foreign bodies in this segment of the eye are close enough to the surface to activate the probe of the electromagnetic locator.[8]

Roentgenographic examination is made for two purposes:

1. To show the presence of a foreign body within the orbital limits, i.e., for diagnosis.

2. To locate accurately the site of the foreign body within or about the globe.

The diagnosis of a foreign body is made by a flat plate postero-anterior view with the patient looking up, then down, as a double exposure; also a flat plate lateral view, double exposure, is made with the patient looking first up, then down. The movement of the foreign body by the two exposures averts confusion with film artefacts that remain fixed and are not present on the other view.

The skeleton-free roentgenographic technic of Vogt is especially indicated to locate foreign bodies in the anterior segment of the eye.[9] In this method the corner edge of a dental film is pressed into the inner canthus and then the roentgenographic beam is directed across the eye from the temple.

If, after complete surgical preparation for removal of the foreign body, the electromagnet, when applied to the eye, produces pain or visible tenting of the iris, it is an indication that a foreign body is in the anterior segment of the eye behind the iris. The application of the magnetic field should be only momentary, and not too strong, or the iris might be injured or the foreign body caught in the iris fibers, making surgical removal more difficult.

Treatment.—Foreign bodies on the lid are removed by surgical antiseptic skin preparation, infiltration local anesthesia with procaine hydrochloride (1 or 2 per cent), and extraction of the foreign body through an incision or the wound of entry, which may be enlarged as necessary.

Foreign bodies in the lacrimal punctum may be extracted with forceps without anesthesia.

Loose foreign bodies on the conjunctiva are removed with a wet cotton applicator. The applicator must be moistened to prevent cotton fibers adhering to the conjunctiva and then acting as another foreign body. Imbedded foreign bodies and concretions of the conjunctiva are removed with a knife point or a sharp spud after a topical anesthetic agent has been instilled. Foreign bodies suspended in the bulbar conjunctiva are grasped under topical anesthesia with sharp-pointed jeweler's forceps, elevated and excised with the points of iris scissors.

After surgical cleansing and topical anesthesia, a foreign body projecting from the surface of the cornea may be touched with a wet cotton applicator or irrigated with physiologic solution of sodium chloride or boric acid solution. If it does not come away easily, no attempt should be made to rub the foreign body off the cornea with the applicator. The choice of a spud or needle point to loosen or extract a foreign body is up to the surgeon.

The edge of the instrument is inserted between the corneal tissue and the foreign body, which is lifted out. The surgeon then curettes any dirt or rust deposit stain from the periphery, working towards the center. If difficulty is encountered in removing the deeper portions of the stain, curettement is stopped, silver nitrate (1 per cent) is applied to the concavity of the ulcer by a finely trimmed applicator, homatropine hydrobromide (4 per cent), and antibiotic ointment instilled, and a firm patch applied. After twenty-four hours the stained portion will be slightly elevated, softened and more easily removed with the golf stick spud or a dental bur.

After removal of the foreign body the patient should be re-examined with the slit lamp to determine whether all the foreign material has been removed and to observe the character and depth of the abrasion produced.

In certain occupations, and sometimes after a blast, inert foreign bodies become imbedded in the cornea and are best left alone if they do not protrude above the surface. Coal particles should be left in the stroma of the cornea. Very frequently, slit lamp examination of coal miners being prepared for cataract extraction has shown many coal particle foreign bodies in the corneal stroma with no sign of irritation.

Deep foreign bodies lying close to Descemet's membrane require special consideration. Even if magnetic they must be loosened from the surrounding stroma before they will respond to the magnetic field. After topical anesthesia is produced, an angular corneal flap is made about the foreign body with a Ziegler discission knife so that the apex of the flap points toward the center of the cornea. After slit lamp examination shows that sufficient depth has been attained, the flap is folded back to expose the foreign body. This is gently freed from the stroma and lifted out. Any rust stain is curetted, and the flap returned to place without suture. An antibiotic and atropine are instilled and the eye firmly patched.

If the foreign body protrudes into the anterior chamber, a discission needle is passed into the chamber from the limbus with the blade held parallel to the iris. The point is gently raised to engage the inner projection of the foreign body while the extraction with forceps is made in the angular corneal flap created as described

in the previous paragraph.[12]

There are two schools of thought encountered in the literature, one advocating that an eye should always be bandaged after simple removal of a foreign body from the cornea, and the second advocating that the eye should be bandaged only in special cases. Text books for years have recommended that the patch be routinely applied. The reasons for bandaging an eye are as follows:

1. Light is excluded, reducing photophobia.
2. The lids are immobilized so that their movement and suction will not enlarge the corneal wound.
3. Infection is prevented by stopping the lid from massaging the contents of the tears deeper into the wound.

Lynch[13] recently gave many reasons why he does not routinely use a patch after simple removal of a foreign body from the cornea. He points out that:

1. Epithelization occurs in a few hours whether or not a patch is worn.
2. The bacterial count increases when an eye is closed with a patch.
3. Hot compresses and local medication cannot be used.
4. Too often the lid is not immobilized, owing to improper application of the dressing.
5. When a patch is applied, the vision is definitely restricted, and binocular vision and its effect on depth perception is removed.
6. Increased loss of time from the job is caused by a general industrial policy of not permitting a man with a patched eye to work.
7. Use of the patch is poor psychology, for the patient's friends and family become unduly concerned over the seriousness of the condition.
8. The application of the patch is a needless, time-consuming expense.

We find that in many simple cases, the patient will do well without a patch, but a doctor should not be dogmatic about either form of treatment. If the eye pad is uncomfortable for the patient, a sensible procedure would be to permit the patient to remove it and to use hot compresses and anodyne medication. If slit lamp examination shows the denuded area to be large or deep with rough edges, and if it appears that the topical anesthesia has softened the corneal epithelium, the patch immobilizing the lid should definitely be used.

A bandage is decidedly contraindicated if purulent dacryocystitis or conjunctivitis is present; also if the patient has only one seeing eye.

Magnetic foreign bodies should be removed from the original wound by applying the magnet over the wound whenever possible.

If the foreign body has penetrated into the anterior chamber, it is attracted to the 12 o'clock position with a hand magnet and extracted through a keratome incision. This site is chosen so that if an iridectomy becomes necessary it will be under the lid. If the foreign body is caught in the iris, or if it is nonmagnetic so that it cannot be attracted to the 12 o'clock position, the keratotomy should be performed at the position of best advantage for a limbal extraction. A nonmagnetic foreign body is grasped with forceps. If the iris prolapses, it is replaced; if it is torn from its base or shredded locally by the procedure, an iridectomy is indicated.

If a magnetic foreign body is behind the iris but in the anterior segment of the eye, the magnet is placed to the opposite side of the pupil to draw the foreign body from behind the iris through the pupil and into the anterior chamber. The magnet is then moved onto the limbus

at 12 o'clock to draw the foreign body into a more favorable position for extraction through a keratome incision.

A nonmagnetic foreign body under the iris is removed by a keratotomy and iridectomy over the foreign body, and a forceps extraction.

A magnetic foreign body on the surface of the lens can be extracted with a hand magnet without causing traumatic cataract. If the foreign body is nonmagnetic, cupped lens forceps may grasp the foreign body and only a localized infiltration may occur, resulting in relatively good postoperative visual acuity. If the foreign body has penetrated the lens capsule, a cataract extraction should be planned. Whenever possible, an intracapsular cataract extraction should be performed; an extracapsular extraction may result in the foreign body's slipping under the iris and becoming lost within the eye. Kirby[14] stated that, even though the zonule is not ruptured, it degenerates with the development of traumatic cataract and the cataract may come away easily by an intracapsular extraction. If the foreign body can be grasped and removed, any type of cataract extraction may follow.

Prognosis.—Since every penetrating ocular wound is potentially capable of causing the loss of an eye, the prognosis at the time of the accident should be guarded. The greatest dangers to visual acuity from foreign bodies located in the anterior segment of the eye are cataract formation and corneal scars over the pupillary area.

Duke-Elder[15] has pointed out that a minute foreign body of a nonirritating type is sometimes responsible for the development of a delayed, chronic glaucoma, the nature of which is missed, unless a careful history is taken and complete examination, including roentgenogram, made. The hypertension may be delayed for months or years from the time of the original accident.

SUMMARY

A foreign body in the anterior segment of the eye may become a serious matter. An outline of a complete examination for diagnosing and locating such a foreign body is presented.

Methods of treatment are developed according to the character and location of the foreign body. In general, the prognosis is good, but there are dangers—traumatic cataract, corneal scars, infection or glaucoma—that may threaten the resultant visual acuity.

RESUMEN

Un cuerpo extraño en la cámara anterior del ojo suele ser una cosa de importancia. Se describe un esquema de examen completo para el diagnóstico y la localización de los cuerpos extraños en la cámara anterior del ojo.

A continuación van descritos los diferentes métodos de tratamiento de acuerdo con la naturaleza y la localización del cuerpo extraño.

El pronóstico por lo general es bueno, pero hay algunos peligros como la catarata traumática, ciactrices corneales, infección o glaucoma que pueden amenazar la capacidad visual resultante.

RÉSUMÉ

La présence d'un corps étranger dans la chambre antérieure de l'oeil peut être grave. L'auteur présente un aperçu de l'examen complet en vue due diagnostic et de la localisation de corps étrangers dans cette partie de l'oeil.

Des méthodes de traitement sont exposées, selon le genre et la localisation des corps étrangers.

Le pronostic est en général bon, mais certains facteurs (cataracte traumatique, cicatrices cornéennes, infection ou glaucome) peuvent présenter une menace pour l'acuité visuelle.

ZUSAMMENFASSUNG

Ein Fremdkörper im vorderen Segment des Augapfels kann ernste Folgen haben. Es wird der Entwurf einer vollständigen Untersuchung zur Erkennung und Lokalisierung von Fremdkörpern im vorderen Abschnitt des Auges vorgelegt.

Der Art und Lage des Fremdkörpers entsprechende Formen der Behandlung werden entwickelt.

Die Prognose ist im allgemeinen gut, doch bestehen gewisse Gefahren, die am Ende zu einer Bedrohung der Sehkraft führen können, wie traumatischer Katarakt, Hornhautnarben, Infektionen oder Glaukom.

RIASSUNTO

Un corpo estraneo nella camera anteriore dell'occhio può essere una condizione grave. Viene descritta la tecnica per una esatta diagnosi e localizzazione.

La cura viene regolata in base ai caratteri e alla sede del corpo estraneo. In generale la prognosi è buona, ma vi è il pericolo della cateratta traumatica, delle cicatrici corneali, delle infezioni e del glaucoma che possono compromettere la vista.

SUMARIO

A presenca de um corpo extranho no segmento anterior do olho, pode causar, complicações de caráter sério. Um plano é apresentado para o diagnóstico e localização de corpos extranhos no segmento anterior do olho. O método de tratamento a ser usado depende do caráter e localização do corpo extranho. Em geral o prognóstico é bom, existindo porém o perigo de complicações tais como "catarata traumática, cicatrízes na córnea, infeções ou glaucoma que podem ameaçar a acuidade visual.

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Diagnosis of the Commoner Glaucomas

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THIS article will discuss the diagnosis of the most important types of adult glaucoma, the so-called primary glaucomas, chronic simple (open angle) glaucoma and acute (angle closure) glaucoma, and several of the most common secondary glaucomas with which they may be confused. These are glaucomatocyclitic crises, pigmentary glaucoma, glaucoma capsulare, acute congestive secondary glaucoma, either uveitic or hemorrhagic, and glaucoma following cataract extraction.

In the ophthalmologist's ordinary routine the history, the visual acuity as measured, and the results of external examination (including biomicroscopic) and gonioscopic) studies, with ophthalmoscopic and tonometric tests will provide most of the basic information and should direct attention to the presence or potential presence of one of the primary glaucomas aforementioned. Other studies depend on this basic information.

History.—In a case of simple glaucoma there may be no symptoms, or there may be mild episodes of ocular pain, headache, blurring vision, colored halos about lighted objects, contraction of the field of vision or even blindness in one eye. In the early stages the symptoms are characteristically unimpressive. On the other hand, patients with acute glaucoma often recount a history that is dramatic, depending on the precipitating factors in each instance and on the presence or absence of congestion. In the noncongestive phase there is blurring of vision or, in a prepresbyopic patient, a decrease in the amplitude of accommodation. Sometimes the onset is associated with rainbow-colored halos or pain in the head or in the eye, particularly at night. The mild symptoms usually last half an hour to two or three hours and then may subside entirely, only to recur at varying intervals, becoming more frequent and continuing longer as time goes on. The circumcorneal injection associated with these episodes is usually slight.

Occasionally the patient states that he finds relief from his symptoms by the use of hot or cold applications, by looking at a bright light for several minutes or by sleep.

Ultimately one of the mild episodes persists longer than usual and suddenly the patient notices marked diminution in vision, photopsia, swelling and redness of the conjunctiva and episclera, and pain in the head and the eyes, often so severe as to cause nausea and vomiting, chills and bradycardia. The sudden change in symptoms often awakens the patient from sleep and occurs without any precipitating factor. It is induced by sudden congestion of the globe, probably a result of the accumulation of tissue metabolites within the eye when the blood supply and drainage of the intraocular fluid is impeded owing to high intraocular pressure.

In some cases the first mild noncongestive episode of acute glaucoma is immediately followed by the congestive phase.

In many patients with episodes of acute angle closure glaucoma the onset of the condition followed a nervous shock. Other important precipitating factors are dilatation of the pupil during dark adaptation,

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as in motion picture theaters; mydriasis for refraction (in predisposed persons); infection about the head, operations on the other eye or about the head, and menstruation.

From the history alone one cannot distinguish between chronic simple glaucoma, glaucomatocyclitic crisis, pigmentary glaucoma and glaucoma capsulare. Similarly, acute congestive secondary glaucoma may resemble acute primary glaucoma so far as history is concerned.

In the case of symptoms similar to those of either of the types of primary glaucoma in a patient with a history of previous cataract extraction, aphakic obstructive glaucoma must be considered.

From the viewpoint of the history alone one must at times rule out such conditions as migraine and recurrent corneal herpes simplex.

Occasionally diagnosis of glaucoma may have been made at some previous time. This may require a great deal of effort to verify.

The *external examination* includes observation of the anterior chamber depth by gross and biomicroscopic means (including gonioscopic evaluation of the depth of the peripheral chamber); biomicroscopic studies of the cornea, aqueous, iris, and lens, and gonioscopic examination. Measurement of the corneal diameter is important, especially for a young patient with acute angle closure glaucoma due to microcornea.

Shallowness of the anterior chamber is usually associated with the characteristic narrowness of the angle in cases of acute angle closure glaucoma, but one must always keep in mind that the anterior chamber may be shallow in the nonglaucomatous eye as well.

Shallowness of the anterior chamber in this form of glaucoma is usually associated with high or relatively high hyperopia, especially during the patient's early adult life.

Biomicroscopic study yields entirely negative in cases of chronic simple glaucoma. When vascular decompensation occurs with acute primary glaucoma, as well as with acute secondary glaucoma, it produces not only chemosis of the conjunctiva but edema of all the ocular tissues. The corneal epithelium is so involved that details of the fundus are obscured. Blebs and vesicles may appear on the cornea. The cornea loses its sensitivity. In the noncongestive stage, even in the presence of high tension, the cornea is usually not edematous, and arterial pulsation is easily seen in the fundus.

In glaucomatocyclitic crises there are a few white mutton-fat deposits on the cornea. There is little or no aqueous flare. In the presence of pigmentary glaucoma a Krukenberg spindle is present on the posterior corneal surface. In cases of capsular glaucoma there may be a deposit of capsular material on the posterior corneal surface. Usually some pigment granules are visible there. In cases of secondary uveitic glaucoma cellular deposits are usually observed on the posterior corneal surface.

The *pupil* is dilated and immobile in both phases of acute glaucoma but is irregular in the congestive phase. The dilatation, when it is not in itself the actual etiologic factor, is probably due to slight stretching of the eyeball and to the pressure effect on the nerves.

In glaucomatocyclitic crises the pupil may be slightly dilated during the attack. In the presence of simple glaucoma it is normal.

The *iris* in the congestive phase of acute glaucoma becomes muddy and discolored. Some of its vessels become visibly distended. If the congestive phase persists for any length of time, fine posterior synechiae may form. In eyes that have become nor-

mal after an acute attack, a whitish scar may result from the localized necrosis in the iris. This atrophy may cause tangential displacement of the iris.

In addition to iris atrophy, one other sign of a previous attack may be present. This is the so-called cataracta glaucomatosa acuta, the Glaukomflecken described by Vogt in 1930.

This form of cataract consists of multiple, circumscribed white spots of characteristic irregular form and radial arrangement lying beneath the anterior lens capsule. Their color varies from the bluish translucency of thin lens opacities to a more porcelain-like whiteness. If seen during an acute glaucomatous attack, the lesions are intensely white. When the intraocular pressure has been reduced their color is much less intense and their transparency increased. The spots have been observed only in the anterior subcapsular cortex, never posteriorly. They do not extend beyond the pupillary zone. None has ever been described as occurring in the region of iridectomy. The axial area is always involved. Here the spots are rounded or dumbbell-shaped and often larger than elsewhere. More peripherally, they are elongated to follow the suture lines of the lens. At the pupillary border where the lesions end, the spots are again often large. In general, they vary from 0.3 to less than 0.02 mm. (Vogt).

These lens opacities do not increase in size with the passage of time. Since they decrease in intensity, however, immediately after the acute epsiode with which they are associated, it is conceivable that the faintest spots may disappear. Once the acute episode is entirely over, the spots do not change in size or color. They do not interfere with the shagreen of the lens. During and immediately after the acute episode the lesions lie just underneath the capsular epithelium. Later, with the formation of new lens fibers, the spots are pushed deeper into the cortex. Vogt observed in 1 case that after five years the opacities were 0.5 mm. within the lens cortex. In 2 cases observed by myself at about the time of the acute episode and again after eight years and six years respectively, the spots occupied a level within the lens cortex equivalent to two-thirds of the thickness of the cornea.

There is no significant visual impairment due to the presence of these thin lens opacities.

In Vogt's opinion the lens opacities are due to exudation. My own view is that they are due to traumatic dissolution of the most delicate terminal portion of the lens fibers at the suture areas, in the uncushioned pupillary area of the anterior cortex. This would account for their radial arrangement. The edema present during the acute attack would account for their more intense whiteness during that time.

Correct diagnosis of Glaukomflecken is important during an acute glaucomatous attack, since the opacities may be mistaken for exudate on the anterior capsule in the presence of marked corneal edema. In other instances a diagnosis of capsular exfoliation may be made.

Recognition of Glaukomflecken may also aid in establishing a definite diagnosis of earlier acute glaucoma in an eye in which an acute attack has been completely relieved. It would be possible also to recognize evidence of two or more previous attacks in the same eye, if the attacks were severe enough to cause the lens lesions and if a sufficient time has elapsed between the separate attacks for new clear lens fibers to form.

In the presence of glaucoma capsulare, capsular material may be present on the pupillary border. The pigment epithelium of the border is usually atrophic. In cases of secondary uveitic glaucoma posterior synechiae are usually present.

Gonioscopic study, in addition to its value in determining the angle depth, may be helpful in some cases of acute angle closure glaucomas in ruling out the angle closure mechanism in diagnosis. In the presence of elevated tension, gonioscopic visualization of the functional posterior portion of the trabecular wall rules out the diagnosis of acute (angle closure) glaucoma. It is the only method of determining the presence or absence of peripheral anterior synechiae, which are pathognomonic of the occurrence of the congestive phase. These synechiae are observed also in cases of aphakic obstructive glaucoma. In many patients with angle closure glaucoma it is difficult to see the angle because of the marked shallowness of the anterior chamber; during a congestive episode it is usually impossible, because of the epithelial edema. In the presence of pigmentary glaucoma the typical dense trabecular pigment ring is always present. With capsular glaucoma a similar ring is present, as well as capsular flecks.

Ophthalmoscopic Study. — The early changes of the optic disc in cases of simple glaucoma are usually impossible to distinguish from physiologic excavation. The cupping is first noted at the temporal side of the disc, with a bend of the vessels at the edge. As the disease progresses one sees the typical picture of a deep cup with an overhanging margin and a pale floor showing the lamina cribrosa. These early changes are recognizable only by comparison of the two discs or by comparison with a previous description of the disc if this is available. If one keeps a routine record of the relative disc and cup sizes in sketch form or as fractions or a ratio of cup-to-disc diameters, the problem is greatly simplified.

The appearance of the disc is one of the important criteria that determine the stage of the disease and thus its prognosis. This is true of both the primary and secondary chronic glaucoma.

In acute congestive episodes of primary or secondary glaucoma the disc usually is not visualized.

Tonometric Study.—From a diagnostic point of view this article is concerned with borderline cases and those in which the tension may even be normal at the time of observation. It is usually unimpressive in the earliest stages of simple glaucoma; one must, therefore, evaluate carefully any suspicious rise in tension. Such a rise in tension, however, will depend on the individual oculist and upon each tonometric calibration. Since establishment of the changes in calibration made by the Committee on Tonometers of the Academy of Ophthalmology, I find my criterion of suspicion of glaucoma with any tension of 26 mm. Schiøtz (1954 scale).

One must be careful in considering a single abnormal tonometer reading, particularly in routine tonometric studies in the office. It should be remembered that a patient's extraocular muscle tonus may greatly influence the tonometric reading. Contraction of the orbicularis may result in a false high tonometric reading, especially if the eye is prominent. I observed a series of 116 normal eyes in my office, taking the tonometric readings with and without the patient voluntarily squeezing his lids together while the tonometer was resting on the cornea and then correlating the result with readings on the Hertel exophthalmometer. There was marked variation, often paradoxic, but the averages indicated a greater tension-raising effect of voluntary lid squeezing in patients with prominent eyes. In 1 patient with thyrotropic exophthalmos, one eye measuring 24 mm. and the other 20 mm., the ability to raise the tension by squeezing the lids was repeatedly greater on the more prominent side. This tends to corroborate the observations on the entire group of 116 normal eyes.

Assuming that suspicion is aroused as to the presence of simple glaucoma, what tests will help to diagnose the condition in early borderline cases? It must be remembered that in such cases visual acuity and the depth of the anterior chamber are normal.

Three aids are available, one subjective, the others objective: (1) studies of the central visual field; (2) tonometric studies, including tonographic procedures, and (3) provocative and other special diagnostic tests.

The earliest change in the visual field in a case of simple glaucoma is accentuation of the angioscotoma tree. This is a reversible change. The next stage is a comma-shaped or sickle-shaped vertical extension of the blind spot, usually above, less frequently below and sometimes both. This is the Seidel sign, which probably has a vascular origin. The Seidel sign is often transient but may become established in association with a slight decrease in the peripheral field limit. This is manifest in the 1/2000 isopter by baring of the blind spot. A small scotoma may occur in the same isopter, unconnected with the blind spot at first. Later it becomes part of the curving extension of the blind spot, which ends in a horizontal line. This is the Bjerrum sign. As the field loss progresses the Bjerrum scotoma meets the peripheral defect to form a Ronne nasal step. The Bjerrum and Ronne scotomas are evidently combined neural and circulatory defects. I need not dwell here on the further field loss, which continues, leaving only central and temporal areas of vision. These, too, may finally be lost.

One must remember that several factors affect visual field determinations materially. These are not only extraocular factors, such as intelligence and attention, but ocular factors, such as myopia, nuclear lens sclerosis and the accommodative and pupil-size effects of miotic drugs. Flicker perimetry gives approximately the same findings as regular scotometry. In equivocal cases it is just as difficult to evaluate. Sometimes it shows changes in the Bjerrum sign area before standard perimetry does.

The central visual field studies within the 30-degree circle are the most important early signs. They have already been discussed.

The visual field in the congestive phase of acute angle closure glaucoma are not usually studied, because of the condition of the patient and his low visual acuity. In the noncongestive phase it is normal or shows a Seidel sign early, but if repeated episodes occur, or if an attack is unrelieved, changes due to pressure may cause changes in the visual field similar to those associated with simple glaucoma.

Tonometric studies are the next step. A difference in tension between the two eyes of more than 3 mm. of mercury may be highly significant. The diurnal curve of ocular tension would be extremely helpful, but it is often impractical to obtain this. It would tend to settle the question whether in the individual case the tension is elevated at a time of day when the oculist is unable, because of practical considerations, to observe it. A variation in the diurnal curve of over 5 mm. certainly should arouse suspicion of glaucoma simplex.

Consideration of the ocular rigidity is sometimes important in borderline cases. As is known, when the tonometer is placed on the eye, the cornea is flattened by the baseplate, the flattened portion is indented by the plunger and the intraocular pressure is raised. The rise in amount of tension produced by a given tonometer load depends on the intraocular pressure plus a number of other factors, the chief of which is the rigidity of the ocular coats. If the eye is more rigid than the average normal eye, the Schiøtz pressure estimate

will be too high, and if it is less rigid, the estimate will be too low.

To determine ocular rigidity one must use two different plunger loads on the tonometer, preferably the 5.5 and 10 Gm. weights. The massage effect of the tonometric procedure is reduced to a minimum by making each application of the tonometer just long enough to obtain a reading. If readings with the 10 Gm. weight consistently are higher than with the 5.5 Gm. weight, then the eye has a higher rigidity than normal and the actual intraocular pressure is lower than either reading. If the reading with the 10 Gm. weight is consistently lower than normal, then the actual intraocular pressure is higher than either reading.

The *provocative tests* are next for consideration. With these one attempts to cause an abnormal elevation of intraocular pressure as evidence of a disturbance in the regulatory mechanism of this pressure.

1. The water test is, in my experience, the most reliable provocative test for simple glaucoma. As the test is generally done, the patient drinks a liter of water within 3 to 4 minutes. No medication should be used during the preceding twenty-four hours. Food and drink should be withheld during the preceding four hours. Tonometric readings are made at twenty-minute intervals for sixty minutes. A rise of 6 mm. of mercury or more (Schiøtz) has been considered evidence of glaucoma. My own interpretation of a positive response to a water test is a rise of over 9 mm. or a rise to 30 mm. or more (1954 scale). The result is positive in approximately 50 per cent of non-surgically treated eyes with known simple glaucoma.

The water test has certain limitations. First, a negative result does not indicate the absence of simple glaucoma. Second, a significant number of tonometric readings of over 27 mm. (1954 scale) occur with normal eyes. These are so-called "false positive" results. Third, the test is not specific for simple glaucoma but may give positive results for other types of glaucoma.

The provocative tests for *acute glaucoma* are based on methods of blocking the chamber angle.

When the pupil is dilated there is an increase in the anteroposterior thickness of the iris at its root. In eyes with narrow angles this leads to further narrowing of the entrance to the angle, permits easier pupillary block and may lead to partial or complete obstruction, with increased intraocular pressure as a result. When this occurs, a definite relation exists between the depth of the chamber angle, the size of the pupil and the thickness of the iris root.

Tests of pupillary dilatation may vary from mere discontinuance of miotic medication in a controlled patient with very shallow anterior chambers to the so-called darkroom test, and finally to the use of mydriatic drugs.

The darkroom test of Seidel is the most valuable as well as the safest provocative test for acute narrow angle glaucoma. It consists of a stay of one to one and one-half hours in a dark room or the use of a light-tight bandage or mask over both eyes for the same period, while the patient remains awake. The tension is measured before and after this test period. A rise of 6 to 8 mm. or more is considered significant.

Clinically, the *mydriasis tests* using drugs carry a certain danger, since occasionally the drug cannot be counteracted, but for investigative purposes these tests are extremely useful.

The drugs that may be used in the mydriasis test for glaucoma are 2 per cent cocaine, 2 per cent euphthalmine, 1 per cent paredrine, 10 per cent neosynephrine and 1 per cent homatropine. The last-

mentioned is, of course, the most dangerous. The tonometric readings are taken ninety to one hundred and twenty minutes after the instillation. An increase of 7 mm. of mercury or more (Schiøtz) is considered evidence of a pathologic condition.

The term *tonography* is used to describe a procedure in which the drop in ocular tension is observed while an electronic Schiøtz tonometer is applied to the anesthetized cornea continuously for four to six minutes. A recording galvanometer is used to record the findings graphically. Medication is stopped for forty-eight hours (longer if DFP has been used). Grant has shown by this method that simple glaucoma is associated with an increased resistance to outflow of aqueous from the eye. The method may make the diagnosis possible when all other conditions at the time are normal, if the coefficient of outflow falls below the normal 0.11. When eyes suspected of being glaucomatous show a coefficient between 0.11 and 0.2, the diagnosis is equivocal, since the normal coefficient is 0.11 to 0.44.

When there is a previous history of glaucoma simplex or when the diagnosis is suggested, if all other studies give negative results tonographic examinations must be done. In my own opinion, the provocative tests, other than the water test, are not of sufficient practical value to use since the introduction of tonography.

The tonographic technic permits evaluation of the effects of treatment, since the treatment causes variation in the flow without affecting the coefficient of outflow. Particularly in cases of self-limited secondary glaucoma, tonographic study permits determination of the time at which the coefficient has returned to normal. It is to be expected that tonographic centers will eventually be established, so that experienced technicians may report their findings to the clinician. Before this happens, however, certain minor changes in technic and data for computation will be expected.

Refractive errors determined while the tension is elevated should not be prescribed but remeasured when the tension is normal.

In the medical follow-up of patients the important diagnostic aids are tests of the visual fields and of tension. These are the criteria by which the efficacy of treatment is usually judged.

Postoperatively one may come up against important diagnostic considerations in which gonioscopic study may be extremely helpful. For example, in 1 patient who has had repeated episodes of acute glaucoma in one eye, relieved intermittently by miotics, and who showed trabecular synechiae, a peripheral iridectomy was successful for only three days. Apparently, enough more of the angle was blocked to require iridencleisis before a successful result was obtained.

Similarly, in 2 instances, peripheral iridectomy accomplished only removal of the anterior portion of the iris as shown gonioscopically, so that no actual communication existed. An acute recurrent attack appeared in each case and was completely relieved by completing the iridectomy.

SUMMARY

The author presents a diagnostic study of the commoner forms of glaucoma, including the acute and "narrow angle" forms. Diagnostic tests mentioned, in addition to the external clinical examination and the anamnesis, including biomicroscopic, gonioscopic, ophthalmoscopic and tonometric studies, as well as examination of the central visual field and various "provocative" and other tests. It is pointed out that important diagnostic considerations may arise after operation has been performed, so that further surgical intervention is required.

RIASSUNTO

Studio diágnostico delle forme più comuni di glaucoma. Oltre all'anamnesi e all'esame obbietiettivo, i mezzi diagnostici citati comprendono studi biomicroscopici, gonioscopici, oftalmoscopici, dell'ampiezza del campo visivo ed altri ancora. Viene sottolineato il fatto che, dopo la terapia chirurgica, possono sorgere importanti problemi diagnostici, così da rendersi necessarii ulteriori interventi.

RESUMEN

El autor presenta un estudio del diagnóstico de las formas comunes de glaucoma, incluyendo las formas agudas y de ángulo estrecho. Además de la exploración externa y de la anamnesis, se mencionan tests diagnósticos, que incluyen estudios biomicroscópicos, gonioscópicos, oftalmoscópicos y tonométricos, así como un examen del campo visual central y de varios otros tests de provocación. Se hace notar que pueden hacerse consideraciones diagnósticas muy importantes después de que la operación ha sido realizada, de manera que requiera una intervención ulterior.

RÉSUMÉ

L'auteur étudie le diagnostic des formes les plus courantes du glaucome. Il cite les divers tests, les examens biomicroscopiques, gonioscopiques, ophtalmoscopiques et tonométriques, ainsi que l'examen du champ visuel central. Il souligne que d'importants problèmes de diagnostic peuvent se poser une fois l'opération exécutée d'où la nécessité de réintervenir chirurgicalment dans certains cas.

ZUSAMMENFASSUNG

Der Verfasser gibt eine Darstellung der diagnostischen Untersuchungen bei den häufigeren Formen des Glaukoms einschliesslich des akuten und des "engwinkligen" Glaukoms. Zu den angeführten Untersuchungen gehören ausser der Erhebung der Krankheitsgeschichte und der äusseren Untersuchung biomikroskopische, gonioskopische, ophthalmoskopische und tonometrische Verfahren und ferner die Prüfung des zentralen Gesichtsfeldes und verschiedene "provokative" und andere Proben. Es wird hervorgehoben, dass wichtige diagnostische Erwägungen nach der Ausführung einer Operation auftreten können und weitere chirurgische Eingriffe erfordern mögen.

Progress is not made by taking pride in our present standards but by critically examining these standards, hypothetically setting higher standards and attempting to achieve them.

—*Rosenstein*

Surgical Treatment of Villous Tumors of the Rectum

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ALTHOUGH the management of adenomatous polyps of the rectum and colon has been standardized to a large extent, many views exist as to the proper surgical treatment of the so-called villous papilloma. It is readily realized that a definite plan of surgical attack is difficult to establish when the surgeon is misled by one or even multiple biopsies.

In comparison, the villous tumor is an infrequent polypoid neoplasm of the rectum and upper colon. Usually it is large, often encircling the entire bowel wall. This growth is characterized by a soft velvety consistency and a broad arrangement of villiform projections on a sessile base. It is pale reddish gray and usually coated with a viscid layer of mucous. The resemblance of villous papilloma to the more frequent neoplastic polyps of the rectum and colon is remarkable except for the diffuse growth pattern observed in the former. This method of growth may well be explained by the wide field of origin of these tumors. This is substantiated by the presence of several pedicles as well as by mucosal changes at the margins of the lesion.

The identifying microscopic features most helpful in diagnosis are:

1. Innumerable elongated fingerlike projections covered with surface epithelium and having a core of connective tissue stroma, small vessels and occasional nerve fibers.

2. The typical goblet cells of mucosa.

3. Normal staining nuclear and cytoplasmic components. In the classic example the histologic pattern is typically benign. There is no evidence of cellular invasion through the lamina propria to the muscularis.

The increased mucoepithelial surface rather than the glandular elements as reflected in the villous configuration illustrates the theory advanced by Dukes that the villous papilloma is the end result of a growth pattern that has affected mainly the superficial epithelium—in contrast to the adenoma, which is the result of a growth pattern affecting mainly cells at a deeper level.

In a series of 32 cases, 42 per cent of the patients were female and 58 per cent male. The average age was 46. Lesions were detected in 2 male patients, aged respectively 17 and 21. In 24 of the 32 cases the lesions were below the peritoneal reflection. Sixteen of the rectal growths were located at the rectosigmoid juncture. The highest lesion occurred in the midtransverse portion of the colon.

In 2 cases there were associated adenomatous polyps. In 4 cases of villous tu-

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mors of the rectum frank adenocarcinoma of the upper part of the colon was present; 2 sigmoidal, 1 in the ascending portion and 1 in the descending portion.

Briefly, the outstanding clinical features consist of slight to profuse discharge of clear or blood-tinged mucus, rectal bleeding and an altered bowel habit, with a tendency to mild diarrhea and protrusion of strands of tissue. No characteristic symptoms differentiate large papillary tumors of the rectum or colon from other large tumors. The quantity of mucus passed by the patient may be greatly increased, but this is not infrequently observed in the presence of other medical or surgical conditions of the colon. Whether this increase in mucus is the result of increased production by the tumor or by the surrounding intestinal mucosa has not been definitely proved.

Some years ago I became impressed by the great propensity of villous tumors toward adenocarcinomatous degeneration. My attention was drawn to the frequency with which serial sections of the excised tumor revealed foci, single or multiple, of malignant change, despite the fact that previous biopsies, the specimens having been taken usually from several sites, gave negative results. Careful observation resulted in positive observations in many cases in which the lesion had a cross-diameter base measuring 3 cm. or more. I also noted that lesions located in the rectosigmoid appeared more reactive than the others and presented a higher percentage of positive signs.

The criteria for the diagnosis of carcinoma consist of (1) anaplasia, characterized by macronucleoli and atypical mitotic figures, and (2) irregularity, featuring intraglandular budding, reduplication of the lumen, true cellular stratification and loss of nuclear and cytoplasmic polarity.

Three types of carcinoma arising in villous tumors are recognized: (1) so-called carcinoma *in situ*, in which anaplasia and irregularity are limited to the glands without invasion of the lamina propia; (2) superficial carcinoma, characterized by anaplasia and irregularity, with reduplication of the lumen and clusters of malignant cells within the lamina propia, but without invasion of the muscularis mucosa and (3) invasive carcinoma, in which there is carcinomatous invasion of the muscularis mucosa and at times vascular or lymphatic extension.

It is my opinion that, except for lesions low in the rectum with a base less than 3 cm., single or multiple, biopsy tissue obtained from selected sites with forceps is not sufficient to establish the diagnosis of a benign tumor. The tumor must be removed *in toto* and serial sectioned. Twenty or more slides should be examined by an experienced pathologist before malignant disease can be ruled out. This extensive microscopic study requires the preparation of 3 mm. serial solitary blocks taken through the entire tumor and part of the bowel wall.

In the 32 cases of villous papilloma studied, malignancy was detected in 18, or 56.2 per cent. In 13 of these the tumors were removed from the rectosigmoid or immediately adjacent sites; in 2 from the middle portion of the rectum, and in 3 from the upper portion of the colon. A large villous tumor, encircling three-quarters of the bowel circumference, was removed by wide segmental resection from the mid-transverse portion of the colon. Examination failed to reveal evidence of malignant degeneration. In comparison to our data, Binkley and Sunderland reported a 68 per cent evidence of malignant degeneration in their series, while more recently Castro and Fisher reported 100 per cent malignancy in 4 cases in which extensive sectioning was done.

In view of my investigation, I am convinced that villous tumors are more ad-

vanced than simple adenomas in the direction of malignancy. It is perhaps true that the transition is more gradual and the tendency for metastasis not so great as with malignant degeneration of an adenoma.

After careful evaluation of my results and those of others, I have adopted the following criteria for villous papilloma of the rectum and colon:

1. Lesions situated 10 cm. or below from the dentate line may be widely removed by electric snare or by direct surgical excision. The latter method is greatly to be preferred if exteriorization by the anal route is possible. After the usual serial sectioning and an intensive pathologic study to determine whether carcinoma *in situ* is present in one area, the patient is kept under monthly observation. If recurrence is discovered abdominoperineal resection is undertaken. In five cases in which this treatment was used there were 3 recurrences classified as Grade III (Broders). The recurrences were all of the adenocarcinoma type, in contrast to the high incidence of the colloid type reported in Binkley's series. If the pathologic report reveals multiple areas of focal malignancy, abdominoperineal resection of the Miles type is immediately carried out.

2. Villous tumors located lower than 10 to 20 cm. are removed by adominoperineal resection, with preservation of the sphincters and without colostomy. If the tumor clinically appears to have invaded the bowel wall and the perirectal or sigmoidal tissue, as determined by induration and immobility, the Miles type of resection is resorted to. In my opinion these tumors harbor carcinoma in either its preinvasive or its invasive state, and radical operation is the most formidable method of attack.

It is realized and accepted that for the aged patients, "poor risks," an operation of this magnitude is not advisable. In such cases multiple snare removal at short intervals may be carried out to control distressing diarrhea, irritability and other discomfort. All surgeons have encountered the difficulty associated with snare excision of large villous tumors in the rectosigmoid and higher in such moribund patients, especially when complicated by an inflamed bowel with a narrow lumen. I am well aware of the possibility of removing all the tumor-bearing surface epithelium at such a high level in a chronically inflamed bowel. The latter condition is frequently coexistent with villous lesions, perhaps as a reactive or irritant type of proctocolitis. The danger of perforation of the bowel wall is forever on one's mind.

3. Villous papillomas of the sigmoid and the upper part of the colon are resected along the same principles as frank adenocarcinoma, irrespective of the size, or consistency of the adjacent bowel wall or the report on frozen sections.

In 1 of my cases of sigmoidal tumor the lesion was relatively small, with a cross-base diameter of 3.5 cm. There was, however, a solitary metastasis to the liver.

SUMMARY AND CONCLUSIONS

All villous tumors must be considered potential harborers of malignant disease.

These growths, although less common than the adenoma and differing in development, have a slightly greater malignant potentiality. In further comparison, the rate of malignant growth and metastasis is slower than that of the true adenomatous polyp with malignant degeneration.

Wide removal of the lesion is advocated in all possible cases.

Lesions with a base measuring 3 cm. or less, located in the lower part of the rectum, may be removed by snare or direct excision. The latter is preferable.

Serial tissue sectioning and intensive pathologic examination of the entire lesion

must be performed before malignant disease can be safely ruled out.

Combined abdominoperineal resection of the Miles type is recommended for small low lesions with multiple malignant foci below 10 cm. from the pectinate line.

Combined abdominoperineal resection of the pull-through type is recommended for villous tumors located approximately 10 to 20 cm. from the pectinate line, unless the tumor has clinically undergone extensive infiltrative malignant changes, in which case the Miles procedure is indicated. In the author's opinion, the recommended procedure allows removal of a greater length of bowel than is permitted by low anterior resection, which admittedly carries the risk of malignant cytologic exfoliation and a greater possibility of recurrence than accompanies a more radical procedure when performed for other than palliative purpose.

Radical segmental resection including 8 cm. of normal bowel distal and proximal to the lesion, together with the avenues of lymphatic and venous spread, is advocated for villous tumors of the upper and more accessible part of the colon.

RÉSUMÉ ET CONCLUSIONS

Toutes les tumeurs à villosités doivent être considérées comme susceptibles de dégénérescence maligne.

Ces excroissances, bien que plus rares que les adénomes et d'une évolution différente, ont une potentialité maligne légèrement plus élevée; leur tendance aux métastases est inférieure à celle des polypes adénomateux.

Il faut autant que possible pratiquer une large excision.

Les lésions dont la base mesure 3 cm. ou moins, situées à la partie inférieure du rectum, peuvent être extirpées à l'anse.

Des coupes en séries s'imposent afin d'exclure avec certitude une lésion maligne.

La résection abdominopérinéale selon Miles, est recommandée lors de petites lésions basses à foyers malins multiples, à moins de 10 cm. du raphé périnéal.

Une résection abdominopérinéale combinée est indiquée lors de tumeurs logées à environ 10-20 cm. du raphé périnéal, à moins de modifications malignes nettes (dans ces cas, opération selon Miles). La technique préconisée par l'auteur permet l'éxércèse d'une plus grande portion d'intestin qu'avec la résection basse.

La résection segmentaire radicale, comprenant 8 cm. d'intestin sain en amont de la lésion, ensemble avec les réseaux lymphatiques et veineux, est recommandée pour les tumeurs à villosités de la partie supérieure et plus accessible du colon.

CONCLUSIONI RIASSUNTIVE

Tutti i tumori villosi devono essere riguardati come potenzialmente maligni. Essi, benchè meno frequenti degli adenomi e diversi nello sviluppo, hanno una maggiore possibilità di trasformazione maligna; tuttavia tale trasformazione è più lenta di quella dei polipi adenomatosi degenerati.

Si deve, in ogni caso, provvedere ad un'ampia asportazione della lesione. Quando sono situati nella parte inferiore del retto ed hanno una base di impianto inferiore a 3 centimetri, possono essere asportati con legature o direttamente, dopo di che è indispensabile provvedere ad un esame anatomopatologico completo, con sezioni in serie, per escludere la presenza di malignità.

Nelle localizzazioni multiple distali, al di sotto dei 10 cm. dalla linea pettinata, viene raccomandata l'amputazione addomino perineale secondo Miles. Per i tumori villosi situati da 10 a 20 cm. dalla linea pettinata si può praticare l'inter-

vento addomino perineale con conservazione, a meno che non siano del tipo infiltrante, nel qual caso si deve fare la Miles.

Questi interventi consentono un'asportazione di intestino maggiore ed evitano il rischio della recidiva.

Anche nei tumori villosi del colon prossimale si raccomandano le ampie resezioni che comprendano almeno 8 cm. di intestino al di sopra e al di sotto del tumor, assieme a tutte le vie linfatiche e venose.

SUMARIO E CONCLUSOES

Todos os tumores vilosos devem ser considerados como séde de degeneração maligna. Esses tumores ainda que menos comuns que os adenômas e diferindo no seu desenvolvimento têm maior potencialidade maligna. Em outra comparação a incidência de transformação maligna e metastases é mais lenta do que aquela dos pólipos adenomatosos com degeneração maligna. Ressecção ampla da lesão é advogada em todos os casos possíveis.

Lesões com base medindo 3 cm. ou menos, localizados no segmento mais baixo recto por coagulação ou excisao directa sendo o ultimo método o de preferencia. O estudo patologico intensivo dos cortes em série, de tôda a lesão deve ser efetuado antes que a degeneração maligna possa com segurança ser descartada.

Ressecção abdomino-perineal combinada do tipo Miles é recomendada para pequenas lesões com múltiplos focos malignos 10 cm. abaixo da linha denteada. Ressecção abdomino-perineal combinada do tipo "pull through" é recomendada para tumores vilosos localizados aproximadamente entre 10 e 20 cms. da linha denteada, a não ser que o tumor ja tenha passado a uma fase de extensa infiltração maligna, o que indicaria o procedimento de Miles. Na opinião do autor, o processo, recomendado permite remover o maíor extensão do intestino do que o permitido pela ressecção anterior baixa, que acarreta o risco de exfoliação de células malignas e uma maior possibililade de recurrência, que a obtida por um processo mais radical quando executado não apenas com intuito paliativo. Ressecção radical do segmento, incluindo 8 centimetros de intestino proximal e distal à lesão, junto com as vias linfáticas e venosas de propagação é indicada para tumores vilosos das porções mais altas e acessiveis do colon.

RESUMEN Y CONCLUSIONES

Todo tumor velloso siempre debe ser considerado como posiblemente maligno. Tales neoplasias, aunque menos comunes que el adenoma y de un desarrollo diferente, som potencialmente algo más malignas. En una comparación más a fondo, el grado de malignidad y la posibilidad de metástasis de estos tumores es menor que en los verdaderos pólipos adenomatosos con degeneración maligna.

Se aconseja la extirpación amplia en todos los casos posibles.

Las lesiones con una base hasta de 3 cm. localizadas en la parte baja del recto, pueden ser extirpadas con el asa de alambre o por excisión directa, preferiblemente lo último.

Es preciso practicar exámenes anatomopatológicos seriados de los tejidos extirpados antes de poder afirmar con seguridad que se trate de un tumor maligno. En los casos de lesiones bajas con múltiples focos malignos y no más lejos de a 10 cm. de la línea pectinada, es aconsejable la resección abdominoperineal combinada tipo Miles. En los casos de tumores vellosos entre los 10 y los 20 cm. de la línea pectinada se aconseja le resección abdominoperineal combinada tipo *Pull-through*, salvo que el tumor haya tomado el tipo infiltrativo maligno extenso en cuyo caso

se debe practicar un Miles. Según la opinión del autor, el procedimiento recomendado pérmite la extirpación de una mayor cantidad de intestino que con la resección anterior, que por otra parte lleva consigo el riesgo de exfoliación citológica maligna y una mayor posibilidad de recidiva.

En los casos de tumores vellosos de una parte del colon más alta y accesible debe practicar una resección segmentaria que incluye 8 cm. por encima y 8 por debajo de la lesión juntamente con vías linfáticas y propagaciones venosas.

ZUSAMMENFASSUNG UND SCHLUSSFOLGERUNGEN

Alle zottigen Geschwülste müssen als Sitz bösartiger Erkrankungen angesehen werden.

Diese Geschwülste sind zwar weniger häufig als Adenome und unterscheiden sich von ihnen in ihrer Entwicklung, haben aber eine grössere Neigung zu bösartiger Degeneration. Ein weiterer Vergleich ergibt, dass das bösartige Wachstum und die Metastasierung der zottenartigen Geschwülste langsamer vor sich geht als bei den echten adenomatösen Polypen mit maligner Entartung.

Die weite Resektion der Erkrankung wird in allen Fällen wo nur möglich empfohlen.

Erkrankungen im unteren Abschnitt des Mastdarms, die eine Basis von drei Zentimetern oder weniger haben, können mit der Schlinge oder mit direkter Resektion entfernt werden. Das letztere Verfahren ist vorzuziehen.

Sorgfältige pathologische Untersuchungen an Serienschnitten des gesamten erkrankten Gebietes müssen ausgeführt werden, um eine bösartige Erkrankung mit Sicherheit auszuschliessen.

Zur Behandlung tief sitzender kleiner Erkrankungen mit mehrfachen bösartigen Herden, die niedriger als 10 cm. von der Anorektallinie liegen, wird die kombinierte abdominoperineale Resektion nach Miles empfohlen.

Die kombinierte abdominoperineale Resektion mit Durchzug des Darmes wird für die Behandlung zottiger Geschwülste, die etwa 10-20 cm. von der Anorektallinie entfernt liegen, empfohlen, es sei denn, dass klinisch eine ausgedehnte bösartige Infiltration nachgewiesen ist; in diesem Falle ist das Milessche Verfahren angezeigt. Der Verfasser glaubt, dass der von ihm empfohlene Vorgang die Entfernung eines grösseren Darmabschnittes ale die tiefe vordere Resektion gestattet, von der man weiss, dass sie die Gefahr der Abschilferung bösartiger Zellen in sich trägt und grössere Möglichkeiten von Rückfällen grössers Möglichkeiten von Rückfällen bietet als das radikalere Verafahren, sofern dieses nicht nur als Palliativbehandlung angewandt wird.

Zur Behandlung zottiger Geschwülste in den höheren und leichter zugänglichen Abschnitten des Dickdarms wird die radikale Resektion eines Darmsegments unter Einschluss von 8 cm. gesunden Darmes in beiden Richtungen vom Sitz der Erkrankung zusammen mit den Lymphbahnen und den Venen, die als Ausbreitungswege in Frage kommen, empfohlen.

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Many Americans who have enjoyed *The Autocrat of the Breakfast Table* and who have been thrilled by a recital of *The Chambered Nautilus* are scarcely aware of the fact that Oliver Wendell Holmes was a physician and for thirty-five years was professor of anatomy at the Harvard Medical School. Medical history does not record any startling anatomical discoveries made by him during this period, but in 1843 he published an article on "The Contagiousness of Puerperal Fever," which remains a classic in medical literature. While Oliver Wendell Holmes's reputation at present rests largely upon his place in American literature, future centuries may reverse this verdict and remember him as the author of a paper on "The Contagiousness of Puerperal Fever," for tastes in literature change with each succeeding generation, while the discovery of a new and great truth remains as fresh as the day it was discovered.

Each age has experimented with pain-killers which have a relation to the dominant medico-surgical interests of the time. Though herbs and the sleeping sponge were still used to some extent, the sixteenth century, the age of anatomy, experimented with such purely anatomical methods for relieving pain as pressure on the carotid artery or other arteries and nerves. The method was in reality a revival of an ancient technique which had long been forgotten. Paré, credulous as he was concerning some of his weird salves, used pressure rather than drugs, a method which also had its dangers and often produced as much pain as it was intended to prevent.

—*Young*

Editorials

International College of Surgeons Forms World Surgical Federation

READERS of *The Journal of the International College of Surgeons*, and of the *Bulletin* that supplements it, are thoroughly familiar with good tidings about the College. On innumerable occasions in the past we have published accounts of new and significant advances in our progress as a world organization, as well as frequent notes—decidedly in a major key—with regard to the increasing success of our efforts toward what has always been our principal goal: primarily, to advance surgery as a science and art and the welding of international friendship and understanding among the world's surgeons as the only safe or desirable means of insuring the world-wide dissemination of surgical knowledge, and thereby providing to all peoples of all nations the highly-trained surgical skill and competence so urgently needed today.

It is our happy privilege to announce, in this issue of the *Journal*, yet another giant step forward. For more than two years the certainty has grown that still closer cooperation among the many Sections of the College is needed and should be furthered by every possible effort. The College has not only progressed, it has grown in membership to some 12,000 surgeons, all of whom have the intellectual, professional and ethical qualifications required of all constituents of the College. It goes without saying that these requirements are high. But this is by no means all; in addition, every member surgeon, whatever his personal ambitions and desires, accepts and supports with all the resources at his command, the prime necessity of united endeavor in every field of work in the world today. It has therefore been agreed that, to strengthen and implement this unity of purpose and action, *a World Federation of General Surgeons and Surgical Specialists* should be created within the International College of Surgeons, each major unit thereof to be autonomous in action and decision and subject to the parent organization only in steadfast adherence to the broad general principles and ideals upon which the College was founded. The final step was taken, after two years of preparation and meetings, during the Congress of the French Section of the International College of Surgeons in Reims on May 23 of this year. At this meeting the culmination of the Federation was a *fait accompli.* The following countries of the newly-formed European Federation were represented: *Austria, Belgium, England, France, Germany, Greece, Italy, the Netherlands, Spain, Switzerland, Turkey* and others.

Four such major units have been established, covering *North America, South America, Europe and Asia.* Since our activities have long since reached a global scale, nothing could be more appropriate. With Sections established in forty of the World's nations and international cooperation among national Sections of the same continent already effectively proceeding by joint voluntary action, we hope and expect to see our many fine scientific Congresses, Federation, national and international, take on new scope and even greater

accomplishment. Moreover, something new will be added: the Federation Congress, to which representatives of all Sections will bring new interest and value every year.

The *European Federation,* which comprises Austria, Belgium, the British Commonwealth, Finland, France, Germany, Italy, the Netherlands, Portugal, Spain, Switzerland and Turkey, will have *Prof. Dr. John Henry Oltramare of the University of Geneva, Switzerland,* as its *Federation Secretary.*

Prof. Dr. Jorge A. Taiana of Buenos Aires, Argentina, will head the *Central and South American Federation,* which consists of Argentina, Bolivia, Colombia, Paraguay, Peru, Venezuela, Costa Rica, El Salvador, Honduras, Haiti, and Nicaragua. Prof. Taiana is former Dean of the School of Medicine and Head of the Department of Surgery, University of Buenos Aires.

Nations making up the *Asiatic Federation* are Burma, Ceylon, China Formosa, China Hong Kong, India, Iran, Israel, Japan, Pakistan, the Philippines, and Thailand. *Prof. Dr. Komei Nakayama, Professor of Surgery at the University of Chiba, Japan,* will serve as Federation Secretary for Asia.

The North American Federation will have as its Secretary *Dr. Curtice Rosser of Dallas, Texas,* President of the United States Section and Professor and Head of the Department of Proctology at Southwestern Medical College. Dr. Rosser will be assisted by *Dr. Ross T. McIntire, Executive Director of the International College of Surgeons.* The nations included in this unit are the United States, Canada and Mexico.

All four Federation Secretaries are not only brilliant and eminent surgeons but dedicated Fellows of the International College of Surgeons. In their hands the new enterprise cannot fail to stimulate growth and activate new ideas on all four continents. Such men have been characteristic of the College since its beginning. Indeed, only such men could have participated so whole-heartedly in the creation of a world-wide surgical fraternity at the time the College was founded, for the idea was unheard of and, like all new ideas, aroused suspicion and antagonism as well as interest. Only such men could have overcome the consequent handicaps, broken down the innumerable barriers of prejudice and envy and brought the College victoriously through to its present impregnable standing in the scientific world. At every such moment as this, when we set up a new milestone of progress and hope, our hearts and minds turn to the memory of those honored and beloved pioneers who are no longer with us—*Fred Albee of New York, Hans Finsterer of Vienna, Konjetzny and Lezius of Germany, Custis Lee Hall of Washington, D. C., Desiderio Roman of Philadelphia, Herbert Acuff of Knoxville, Tennessee,* and others whose names there is not space for here but whose loyalty and dedication have made those names luminous forever. One cannot help longing, at such a time, for the power to set aside time and change and call them back to see the rich fruition of their labors. Many tried and tested Fellows of long standing, thank God, are still with us and enjoy this privilege.

It would be impossible to trace, through the intricate and widespread activities of the College today, the origin of each of the brilliant ideas upon which those activities were founded. One thing, however, is certain: no such idea emerged from a second-rate mind. While the second-rate minds occupied themselves, as is their wont, with purposeful self-aggrandizement at the expense of others, our Fellows have thought of others before themselves and have not balked at sacrifice or hardship when their service to the College and to mankind required it of them. "Gentlemen unafraid" pretty well describes both

them and their successors. We congratulate Drs. Rosser, Taiana, Oltramare and Nakayama on their well-merited inheritance of this honored tradition, and we congratulate the College—and the world—on their unhesitating acceptance of the responsibilities they have assumed. While such men are still to be found, the College will continue to surmount every hurdle set across its path either by nature or by design, for eventual laurels are the reward of the steadfast.

M. T.

The Twenty-Second Annual Congress

of the

International College of Surgeons

In Connection With

Harvey Tercentennial

(List of essayists incomplete)

As we go to press, plans are being crystallized for the Twenty-Second Annual Congress of the United States and Canadian Sections of the International College of Surgeons to be held at the Palmer House in Chicago, September 8-12, 1957.

This year the medical world celebrates the Tercentennial of William Harvey, discoverer of blood circulation. As a part of the Congress program there will be special ceremonies at the International Surgeons' Hall of Fame to commemorate the occasion. On September 11 a wreath will be placed before the statue of Harvey in the Speidel Hall of Immortals. This act will be a symbol of the continuity and universality of science—both aims of the College. In keeping with this theme, **Professor Dr. Leopold Schoenbauer,** Chairman and Head of the Department of Surgery, University of Vienna, and President of the Medical Historical Society of Vienna, will give an address on the Harvey Tercentennial occasion.

There will be many other outstanding Congress participants. The scientific program is under the general chairmanship of **Dr. Raymond W. McNealy** of Chicago, and his co-chairman is **Dr. Richard M. H. Power** of Montreal, Quebec. **Dr. Ross T. McIntire** is Executive Director. Space does not permit a complete listing of all the speakers who will come, not only from the United States and Canada but also from many other nations; however, a partial roster of those who will participate in the various sessions appears below.

Arthur C. Allen, Associate Professor of Pathology, Sloan-Kettering Division, Cornell University Medical College, New York—*The Current Status of the Clinico-Pathologic Correlation of Pigmented Nevi and Malignant Melanomas*

Lyon H. Appleby, Associate Professor of Surgery, University of British Columbia, Vancouver—*Current Concepts of Surgical Management of Peptic Ulcer*

Harry E. Bacon, Professor and Head of Department of Proctology, Temple University School of Medicine, Philadelphia.

M. A. van B. Bastiaanse, Wilhelmina Hospital, Amsterdam, The Netherlands—*Vesicovaginal Fistulae*

Mario Battezzati, Director, Institute of Pathologic Surgery, University of Parma, Parma, Italy—*The Ligature of the Internal Mammary Arteries in the Surgical Treatment of Coronary Insufficiency*

Claude S. Beck, Professor of Cardiovascular Surgery, Western Reserve University School of Medicine, Cleveland—*Surgical Operations for Coronary Artery Disease—New Physiology*

I. Boerema, Professor of Surgery, University

of Amsterdam Medical School, The Netherlands—*Total Gastrectomy*

Wilbrod Bonin, Dean of the Faculty of Medicine, University of Montreal, Montreal, Quebec—*Embryologic Problems in Relation to Surgery*

Robert J. Booher, Assistant Professor of Clinical Surgery, Cornell University Medical College, New York—*Current Concepts in the Management of Carcinoma of the Stomach*

Paul Bucy, Professor of Surgery, Northwestern University Medical School, Chicago

Louis A. Buie, Emeritus Professor, Proctology, University of Minnesota Graduate School, Rochester, Minnesota—*Fistula in Ano*

Daniel Callahan Jr., Professor of Urology, Cook County Post Graduate Medical School, Chicago—*Urologic Complications in Gynecologic Surgery*

John Charnley, Orthopedic Surgeon, University of Manchester, Royal Infirmary, Manchester, England

Raymond Darget, Professor of Urology, University of Bordeaux, France—*Treatment of Bladder Tumors*

Michael R. Deddish, Associate Professor of Clinical Surgery, Cornell University Medical College, New York—*Lymphadenectomy at Time of Resection of Distal Colon and Rectum—Five Year Results*

Frederick A. Figi, Senior Consultant, Section on Plastic Surgery and Laryngology, Mayo Clinic, Rochester, Minnesota—*Diagnosis and Management of Malignant Tumors of the Nasal Fossae and Accessory Sinuses*

Edgar L. Frazell, Assistant Professor of Surgery, Cornell University Medical College, New York—*Surgical Treatment of Cancer of the Tongue*

Alfonso de la Fuente, Professor of Surgical Pathology, Faculty of Medicine, University of Madrid, Spain—*Thoracic Surgery*

Masao Fujimori, Instructor, Kimoto's Surgical Department, School of Medicine, Tokyo University, Tokyo, Japan—*Morphological, Biochemical and Endocrinological Studies of Breast Cancer*

Carlos Gama, Professor of Neurosurgery, Faculty of Medicine, University of Sao Paulo, President, International College of Surgeons, Sao Paulo, Brazil—*Topic to be announced.*

John H. Garlock, Clinical Professor of Surgery, Columbia University College of Physicians and Surgeons, New York—*The Surgical Management of Ulcerative Colitis*

Edwin J. Grace, Instructor of Surgery, Long Island Medical College, Brooklyn—*Uses and Misuses of Antibiotics in Surgery*

Francisco Graña, Former Head of Department and Professor of Surgery, San Marcos University, Lima, Peru—*The Predecessors of Harvey*

Werner Hagmaier, Chief Surgeon, Stadt, Krankenhauser, Schopfheim/Baden, Germany—*Lumbar and Subdiaphragmatic Aortography*

John P. Heaney, Assistant Clinical Professor of Surgery, Baylor University and University of Texas Post Graduate Division, San Antonio—*Chest Injuries*

George W. Holmes, Professor of Thoracic Surgery, Cook County Graduate School of Medicine, Chicago—*Current Concepts of Pulmonary Tuberculosis*

Marc Iselin, Surgeon, American Hospital, Paris, France—*Current Concept of Surgery of Tendons*

Chevalier L. Jackson, Professor and Head of Department of Laryngology and Bronchoesophagology, Temple University School of Medicine, Philadelphia—*The Role of the Endoscopist in the Management of Chest Problems*

Ronald Jarman, Vice President, Association of Anesthetists of Great Britain and Ireland, London, England—*The Anesthetist and His Position with the Surgical Team*

Victor Kremens, Assistant Professor, Radiology, Hahnemann Medical College, Philadelphia—*Roentgenography of the Breast*

Henri Laborit, Director, Experimental and Clinical Research on Artificial Hibernation, Army Research Center, General Military Hospital, Val de Grace, France—*A New Anesthetic Agent* and *The Clinical Application of Artificial Hibernation and Potentialized Anesthesia*

John S. Ladue, Assistant Professor of Medicine, Cornell University Medical College, New York—*The Evaluation and Preparation of Poor Risk Patients for Major Surgery*

Claude Lambert, Associate Professor of Orthopedic Surgery and Acting Head of Department, University of Illinois College of Medicine, Chicago—*The Doctor and the Amputee Problem*

J. Edouard Lemieux, Notre Dame de Ste Croix Hospital, Mont-Laurier, Quebec—*Hernias*

G. Kenneth Lewis, Clinical Associate Professor of Otolaryngology, University of Illinois College of Medicine, Chicago—*Electri-*

cal Burns

Lloyd G. Lewis, Professor, Clinical Urology, Georgetown University School of Medicine, Washington, D. C.—*Diagnosis of Cord Bladder*

Manuel E. Lichtenstein, Professor of Surgery, Cook County Graduate School of Medicine, Associate Professor of Surgery, Northwestern University Medical School, Chicago —*Hyperthyroid Pyloric Stenosis in Adults*

Jacob J. Longacre, Associate Surgeon, Research and Attending Plastic Surgeon, Cincinnati General Hospital, Cincinnati—*Reconstruction of the Head and Neck Following Radical Surgery for Carcinoma*

Raymond W. McNealy, Emeritus Professor of Surgery, Northwestern University Medical School, Chicago

Newton Mead, Assistant Professor of Orthopedic Surgery, Northwestern University Medical School, Chicago

Oliver Monod, Founder of the French Society of Thoracic Surgery, Paris, France—*Hypothermia in Cardiopulmonary Surgery*

Juan Netto, Professor of Clinical Surgery, Faculty of Medicine, and Chief, Thoracic Surgery Department, Ascuncion, Paraguay —*Pulmonary Cysts in Newborn*

Walter L. Palmer, Richard T. Crane Professor of Medicine, University of Chicago—*The Role of Corticotropin and Adrenal Steroid Hormones in the Treatment of Chronic, Nonspecific Ulcerative Colitis*

Philippe M. Polani, Chief Radiologist, l'Hôpital d'Israelite, Alexandria, Egypt—*Radiological Aspects of Duodenal Ulcers*

Gerald H. Pratt, Associate Clinical Professor of Surgery, New York University College of Medicine, New York—*Selection of Patients and Types of Grafts in Aortic and Arterial Replacement* and *Extracorporeal Circulation for Complete Bypass of the Heart and Lungs in Open Cardiac Surgery*

Charles B. Ripstein, Professor of Surgery and Executive Officer, Department of Surgery, Albert Einstein College of Medicine, Yeshiva University, New York — *A New Concept in the Surgical Management of Bleeding Esophageal Varicoses*

Louis P. River, Professor of Surgery, Cook County Graduate School of Medicine, Chicago—*Management of Benign Tumors of the Breast*

Esteban Rocca, Professor of Neurosurgery, San Marcos University, Lima, Peru—*Surgical Treatment of Neuro-Cysto Sarcosis*

Curtice Rosser, Professor and Head of Department of Proctology, Southwestern University Medical College, Dallas

A. K. Saha, Hunterian Lecturer, Royal College of Surgeons of England, Professor of Surgery, Nibratan Sircar Medical College, Calcutta, India

Shigeru Sakakibara, Professor of Surgery, Tokyo Women's Medical College, Tokyo, Japan—*Intracardiac Surgery with Improved Heart-Lung Machine—Studies on the Cardioscope*

Leopold Schoenbauer, Chairman, Department of Surgery, University of Vienna, Austria—*Address for Harvey Tercentennial*

Alvin O. Severance, Professor of Pathology, Baylor University College of Medicine, San Antonio—*Pathology of Benign Tumors of the Breast*

José Soler-Roig, Professor of Surgery, University of Barcelona, Spain—*The Importance of Routine Cholangiography in Gallbladder Operations*

Orion H. Stuteville, Professor Maxillofacial Surgery, Northwestern University Dental School, Lecturer in Surgery, Northwestern University Medical School, Chicago—*Lesions of the Oral Cavity: Infections of the Head and Neck*

Jorge Taiana, Former Professor of Surgery and Dean of Medical Faculty, University of Buenos Aires, Argentina—*Hydatid Cysts of the Lung* and *Caneer of the Lung, Late Results—Five Year Survival*

Gershom J. Thompson, Professor of Urology, Mayo Foundation, University of Minnesota, Rochester—*The Surgical Management of Neurogenic Cord Bladder*

Kazumi Taguchi, Assistant Professor of Surgery, Chief of Cardiac Division, Okayama University Medical School, Okayami-shi, Japan—*Open Cardiac Surgery Under Selective Brain Cooling*

Arthur M. Vineberg, Lecturer in Surgery, McGill University Medical School, Montreal, Quebec—*The Treatment of Coronary Artery Insufficiency by Internal Mammary Artery Implantation—Seven-Year Study*

Harold Voris, Professor of Neurosurgery, Stritch School of Medicine, Loyola University, Chicago

James Watts, Professor of Neurological Surgery, Head of Department of Neurology and Neurological Surgery, George Washington School of Medicine, Washington, D.C.

William L. Watson, Professor of Clinical Surgery, Cornell University Medical College, Associate Professor of Surgery and

Director of Cancer Coordination, New York University Bellevue Medical Center, New York—*Esophageal Carcinoma*

Charles Stanley White, Head of the Department and Professor of Surgery, George Washington University, Washington, D.C.—*Clinical Research in Malignant Metastases*

Stimulating Section Programs have been planned—many of them joint sessions. The incomplete schedule follows:

Neurologic Surgery — Chairman, **James W. Watts,** Professor of Neurological Surgery and Head of the Department of Neurology and Neurological Surgery, George Washington University School of Medicine, Washington, D. C.

Tuesday afternoon and evening. Joint with General Assembly, Monday afternoon

Obstetrics and Gynecology—Chairman, Honorary, **Virgil S. Counseller,** Professor of Surgery, Mayo Foundation, University of Minnesota Graduate School of Medicine, Rochester, Minnesota; Chairmen, **J. P. Greenhill,** Professor, Cook County Graduate School, Chicago; **Emmet A. Pearson,** Senior Staff, California Hospital, Los Angeles; **August F. Daro,** Professor of Obstetrics and Gynecology, Cook County Post Graduate School of Medicine, Chicago

Monday, Tuesday and Wednesday afternoons. Joint with General Assembly, Thursday afternoon

Ophthalmology and Otorhinolaryngology—Chairman, Ophthalmologic Surgery, **Girolamo Bonaccolto,** Clinical Professor of Ophthalmology, New York University College of Medicine, New York; Chairman, Otolaryngologic Surgery, **Alan Austin Scheer,** Assistant Clinical Professor of Otolaryngology, New York University College of Medicine, New York

Monday, Tuesday and Wednesday, all day. Otolaryngologic—Joint with General Assembly, Tuesday morning

Orthopedic Surgery—Chairman, **Arthur Steindler,** Professor of Orthopedic Surgery, State University of Iowa, College of Medicine, Iowa City, Iowa

Tuesday and Wednesday afternoons. Joint with General Assembly, Monday afternoon

Plastic and Reconstructive Surgery—Chairman, **Neal Owens,** Clinical Professor of Plastic and Reconstructive Surgery and Head of Department, Tulane University School of Medicine, New Orleans

Tuesday and Wednesday afternoons. Joint with General Assembly, Monday afternoon

Coloproctologic Surgery—Chairman, **Harry E. Bacon,** Professor and Head, Department of Proctology, Southwestern University Medical College, Dallas

Tuesday and Wednesday afternoons. Joint with General Assembly, Tuesday afternoon

Urologic Surgery — Chairman, **Lowrain E. McCrea,** Clinical Professor in Urology, Temple University School of Medicine, Philadelphia

Monday and Tuesday afternoons. Joint with General Assembly, Wednesday, afternoon

Surgical Nurses — Chairman, **Margaret Young,** Chicago

Monday and Tuesday, all day

Inhalation Therapists—Chairman, **Lawrence A. Fruik,** Chicago

Thursday, all day

On Monday evening the Film Forum will be held in the Grand Ballroom of the Palmer House. Chairman of the Forum is **Dr. Philip Thorek,** Associate Professor of Surgery, University of Illinois and Cook County Graduate Schools of Medicine, Chicago. Moderator for the evening will be **Dr. Max Thorek,** Professor of Surgery, Cook County Graduate School of Medicine and Founder and International Secretary General, International College of Surgeons. Speakers and films for the program are as follows:

Jorge Taiana, Former Professor of Surgery and Dean of the Medical Faculty, University of Buenos Aires, Argentina—*Hydatid Cysts of the Lung*

I. Boerema, Professor of Surgery, University of Amsterdam, Medical School, The Netherlands—*Total Gastrectomy*

Henri Laborit, Director, Experimental and Clinical Research on Artificial Hibernation, Army Research Center, General Military Hospital, Val de Grace, France—*The Clinical Application of Artificial Hibernation and Potentialized Anesthesia*

Another highlight of the Congress will be the unveiling of a portrait of Crawford Long, a gift of the Georgia Chapter of the

International College of Surgeons. Still other highlights will include the Officers and Regents' Luncheon on Sunday, September 8, at 12:30 P.M. in the Crystal Ballroom of the Palmer House, and on Thursday, September 12, at 7:00 p.m., **Senator Hubert H. Humphrey** of Minnesota will deliver the Convocation Address at the Civic Opera House. The Woman's Auxiliary, under the leadership of **Mrs. Clifton L. Dance,** President, has arranged lectures, teas and special events for the wives of the doctors attending the Twenty-Second Annual Congress.

For further information about the Program, write:

Dr. Ross T. McIntire, Executive Director
International College of Surgeons
1516 North Lake Shore Drive
Chicago 10, Illinois

PRELIMINARY SCHEDULE

Advance Registration—Sunday, September 8, 10-12 a.m., 2-5 p.m., Fourth Floor, Palmer House

Registration—Monday, Tuesday and Wednesday, 8:30 a.m.—5 p.m.; Thursday, 8:30 a.m.—3 p.m.

Meetings of the United States and International Executive Councils and Board of Trustees, United States Section, Sunday, morning and afternoon

Postgraduate Instructional Courses—Orthopedics, Sunday morning and afternoon; Ophthalmology and Otolaryngology, Sunday afternoon

General Assemblies—Two Sections, Monday through Thursday mornings; One Section, Monday through Wednesday afternoon; Two Sections Thursday afternoon

Film Forum—Monday, 8-10 p.m.

Officers and Regents' Luncheon—12:30 p.m. Sunday

Special Events—Includes Harvey Memorial Ceremony, Tuesday afternoon, Hall of Fame

Canadian Section—Business meeting, 5 p.m.; dinner 6:30 p.m., Tuesday

Social Hour and Annual Banquet—Wednesday, 6-10 p.m.

Scientific Motion Picture Program—9 a.m. to 5 p.m. daily

Woman's Auxiliary—Program daily

Section Programs:

Coloproctologic Surgery—Tuesday morning, organizational meeting; Tuesday afternoon, section meeting, 1:30-3:00; joint meeting with General Assembly 3:30-5:00; Wednesday 1:30-5:00

Inhalation Therapy—Thursday morning and afternoon

Neurologic Surgery—Monday with Orthopedic and Occupational Surgery and General Assembly 3:30-5:00; Tuesday section meetings morning and afternoon

Obstetrics and Gynecology—Monday, Tuesday and Wednesday afternoons; Luncheon, Wednesday; joint meeting with General Assembly 1:30-3:00 Thursday

Occupational Surgery—Joint meeting with General Assembly Monday afternoon and Tuesday morning

Ophthalmology and Otorhinolaryngology—Monday, Tuesday, and Wednesday morning and afternoon; luncheon Wednesday; Joint session for Otolaryngology Tuesday morning with General Assembly

Orthopedic Surgery—Monday 3:30-5:00 p.m., joint meeting with Neurological Surgery and General Assembly; Section meetings Tuesday and Wednesday afternoons

Plastic and Reconstructive Surgery—Tuesday morning with General Assembly; Section meetings Tuesday and Wednesday afternoons

Surgical Nurses' Section—Monday and Tuesday morning and afternoon

Urological Surgery—Monday and Tuesday afternoon; joint meeting with General Assembly, Wednesday afternoon

New Books

Books Received.—The following books have been received by the Editor; they will be reviewed critically as space and facilities permit. Omission of more extended review, however, is not to be taken as criticism of the merit of the book.

Operative Surgery. Edited by Charles Rob and Rodney Smith. London: Butterworth & Co., Ltd.; Philadelphia, F. A. Davis Co., 1956. Vols. 1 and 2 of 8 (plus index). Profusely illustrated.

Proceedings of the Third National Cancer Conference, Detroit, Michigan, June 4-6, 1956. Sponsored by the American Cancer Society, Inc., and National Cancer Institute, U. S. Public Health Service. Philadelphia: The J. B. Lippincott Company, 1957. Pp. 961.

Principles of Urology. By Meredith F. Campbell. Philadelphia and London: The W. B. Saunders Company, 1957. Pp. 566, with 319 illustrations. *Reviewed in this issue.*

Le Diagnostic du Cancer d'Estomac a la Periode Utile (Diagnosis of Carcinoma of the Stomach at the Time Most Favorable for Treatment). By Rene A. Gutmann. Paris: G. Doin et Cie, 1956. Pp. 257.

Pathology and Surgery of the Veins of the Lower Limbs. By Harold Dodd and Frank Cockett. Baltimore: The Williams & Wilkins Company, 1957. Pp. 462, illustrated.

Physiologic Principles of Surgery. By Leo M. Zimmermann and Rachmiel Levine. Philadelphia: The W. B. Saunders Company, 1957. Pp. 988. *Reviewed in this issue.*

Spinal Cord Compression. By I. M. Tarlov. Springfield, Ill.: Charles C Thomas, Publisher, 1957. Pp. 147, with 41 illustrations.

Muscle Relaxants in Anesthesiology. By Francis F. Foldes. Springfield, Ill.: Charles C Thomas, Publisher, 1957. Pp. 210, with 9 illustrations.

Brain Mechanisms and Drug Action. By William S. Fields, Springfield, Ill.; Charles C Thomas, Publisher, 1957. Pp. 167, with 69 illustrations.

The Clinical Management of Varicose Veins. By David Woolfolk Barrow. New York: Paul B. Hoeber, Inc., 1957. Pp. 167, with 70 illustrations.

Head Injuries and Their Management. By Francis Asbury Echlin. Philadelphia: The J. B. Lippincott Company, 1956. Pp. 127, with 10 illustrations.

Principles of Urology. By Meredith F. Campbell. Philadelphia and London: The W. B. Saunders Company, 1957. Pp. 566, with 319 illustrations.

The author has previously written two books on urology: *Clinical Pediatric Urology*, a guide to the management and treatment of urologic diseases in children, and the three-volume *Urology*, a mine of complete and precise information in the urologic field. Now comes a third textbook, primarily for the instruction of medical students in the broad fundamental principles of Urology and to serve as a guide for the general physician who is not a urologist.

The first chapter, on urologic semantics, is most instructive. In roentgenographic examination a roentgen film, not a roentgen plate, is used. The preliminary exposure may be a plain or scout film. Intravenous urographic study should properly be called excretory urographic study. The solution used for injection is a medium, not a dye. These are just a few examples of the proper use of medical terms as set forth in this chapter.

The etiology, pathology, symptoms and diagnosis of diseases of the genitourinary tract are discussed in detail. Technics of various operations are covered by references to the author's previous works. The chapter on embryology and anomalies of the urogenital tract is comprehensive, occupying 90 pages of the excellent text.

At the end of the book there are 19 pages of questions that may be asked of students, covering the material in each successive chapter.

Beautifully arranged and beautifully written, this book should be invaluable for teaching medical students the fundamental principles of urology. For the non-urologist who desires a better understanding of diseases of the urogenital tract this book cannot fail to serve an excellent purpose.

BERNARD E. COHLER, M.D.

Physiologic Principles of Surgery. By Leo M. Zimmerman and Rachmiel Levine. Philadelphia: The W. B. Saunders Company, 1957. Pp. 988.

A book written by more than one author can be praiseworthy if properly edited. The excellent editing associated with this book, plus brief but worth-while editorial notes, makes it outstanding. No one person is omniscient, and the collaboration of surgeon and physiologist is a necessity today. Fortunately the practical aspects of clinical surgery have not been minimized in this erudite presentation.

For special commendation must be mentioned Chapter 28, which deals with the adrenal glands. Another outstanding chapter is Chapter 26, which discusses the thyroid gland.

This book can be recommended to anyone interested in clinical surgery or the more theoretical phases included in the physiologic principles.

PHILIP THOREK, M.D.

The Principles and Art of Plastic Surgery. In 2 volumes. By Harold Gilles and R. Ralph Millard Jr. Little, Brown & Company, 1957. Pp. 652.

In this human and readable two-volume work the authors, in a rather unorthodox style, trace the development of plastic surgery in England from the early part of World War I to the present. Almost every aspect of plastic and reconstructive surgery is covered. There are numerous illustrations of technics formerly used, as well as the most up-to-date methods. Each principle is illustrated by a short personal case history. It would be a monumental task to give a detailed review of the 600 pages of this work.

As the first few patients began arriving at Aldershot in 1915, it became evident that "normal tissue should be returned to normal position." This was well illustrated by Private

Bell, who arrived with a small mouth that opened vertically. After the scar was excised the nose and the remaining lip tissue sprang back into normal position, and the defect was ultimately filled with a cheek flap. The result was creditable.

The advantages and disadvantages of local flaps, V to Y incisions, caterpillar flaps, fat and muscle flaps were soon demonstrated. As the early hurdles of bone grafting, lack of nasal linings and lack of prostheses were conquered, the value of plastic surgery was recognized, and the plastic unit was transferred to larger quarters at Sidcup in Kent. With many casualties among the Dominion troops, the Colonial units were combined at Sidcup.

On October 3, 1917, Gillies formed his first tube pedicle, only to find that Filatoff had forestalled him in the publication of this technic. After the initial use of the tube there was much enthusiasm and rapid development of this method of repair. As the war progressed, simplifications and improvements of older methods grew out of constant experimentation. Esser's inlay principle was improved and simplified. Many problems, such as nasal loss and contracted or sunken eye-sockets, were met and conquered. Final touches on the remaining faces kept Sidcup busy for several years after the war. After World War I plastic surgery entered a period of transition during which it was recognized as a specialty.

An interesting chapter by Ivan Magill describes the many problems of anesthesia and the development of various refinements therein.

The chapter on "technical tips" points out the value of many useful technical details the authors developed from years of experience. Treatment of scars, wound care, suturing, keloids and many other topics are covered. Split skin grafting is quick, simple and often all that is necessary for many wounds. Absolute fixation and pressure are essential for good "take."

Chapters devoted to rotation flaps, direct flaps, tube pedicles and forehead flaps describe in detail the application, technic, dangers and safeguards of these useful methods.

Internal and external radiation has been found most useful in the treatment of hemangiomas. Operation is frequently necessary as primary treatment and in the revision of secondary scars due to irradiation. Radiation burns were common in early years of roentgen and radium treatment. The characteristics of roentgen burns and the indications for the excision are given. Excision is followed by primary closure or the application of a skin graft or a pedicle flap.

The treatment of carcinoma should be a team effort with a split purpose—cure first, repair second. In the authors' opinion, reconstruction should not be delayed because a recurrence may develop.

Reconstruction of the ear is far from perfect, but, because of its psychic effect, it should be continued. The problems involved in the various methods of otoplasty are described. Various technics for repair of harelip and cleft palate are discussed. With cleft palate the real problem of lack of tissue has not been solved, and the old principle of the tube pedicle may be part of the answer.

Cosmetic surgery is extremely useful, changing the entire psychic outlook of many patients. Principles evolved from traumatic and reconstructive surgery are applicable to cosmetic surgery. World War II brought more problems and more progress. The need of proper early treatment and early evacuation was solved by the formation of surgical field units and special evacuation technics that brought many a wounded soldier to the Base Hospital in a matter of hours.

The subject of burns is adequately covered, with emphasis on early skin grafting and reconstruction. Chapters are devoted to reconstruction of the lip, and eyelid, as well as to facial fractures.

The final chapter, "A Day in the Clinic," is typical of the entire text, leaving one with the feeling of actually knowing the patients with their defects and problems.

These books are recommended not only to the plastic surgeon but to all medical personnel, as they offer a combination of practical and academic information in addition to hours of pleasant reading.

RICHARD W. HUGHES, M.D.
AND
NEAL OWENS, M.D.

Abstracts from Current Literature

Changes in the Bronchial Epithelium in Relation to Smoking and Cancer of the Lung. Auerbach, O., Gere, J. B., Forman, J. B., Petrick, T. G., Smolin, H. J., Muehsam, G. E., Kassouny, D. Y., and Stout, A. P., New England J. Med. 256:97, 1957.

The bronchial epithelium from 119 cadavers was studied to determine whether inhalants produced hyperplasia, metaplasia and neoplastic changes in heavy smokers who died either from bronchogenic carcinoma or from some other diseases. Basal cell hyperplasia, squamous metaplasia and carcinoma in situ were least frequent in those who had never smoked. The degree of change in the epithelium paralleled the increased consumption of cigarettes. These results are consistent with the theory that cigarette smoking is an important factor in the causation of bronchogenic carcinoma.

ERNEST G. DEBAKEY, M.D.

A Study of Motility in the Gastric Remnant Following Subtotal Gastrectomy. Jordan, G. L. Jr., Barton, H. L., and Williamson, W. A., Surg., Gynec. & Obst. 104:257, 1957.

Forty-six patients were studied between the ninth and fifteenth postoperative days by roentgenographic examination of the stomach and by measurement of the intraluminal pressure.

In the intact stomach, the rate of evacuation is influenced by antral tone and strong antral contractions, which may exceed 100 mm. of mercury in force, as other investigators have indicated. In the fundus and cardia there is moderate persistent tone, but peristaltic waves are infrequent and feeble and contribute little to the propulsion of food.

The capacity of the gastric remnant varied from patient to patient; all, however, were able to ingest at least 10 ounces of barium. The time required for complete gastric emptying varied from five minutes to more than seven hours, excluding a patient with efferent loop obstruction. There were 6 asymptomatic patients in whom complete emptying of the stomach required more than three hours. There was a significant difference in the emptying time of patients who had gastroduodenostomies as compared to those who had gastrojejunostomies. After a gastroduodenostomy, the median emptying time was two hours, with a mean emptying time of one hundred and sixty-nine minutes. After a gastrojejunostomy, the median was sixty minutes and the mean seventy-six minutes.

In 8 patients studied in the Trendelenburg position, emptying took twice as long and, in 1 instance, four times as long. In 2 patients, however, the emptying time was actually shorter with the patient in the Trendelenburg than in the upright position.

The recordings of intragastric pressure were characterized by a minimum of motility. In some patients, no gastric contractions were observed during the entire period of observation. In no instance did the intraluminal pressure exceed 15 to 20 mm. of mercury. Patients with efferent loop obstruction demonstrated the greatest amount of motility; all contractions, however, were feeble.

The motility in the gastric remnant is minimal in the immediate postoperative period and is not significantly different from that which occurs in this portion of the intact stomach. The emptying of the stomach does not depend upon propulsive movements; therefore, gastric atony is not a primary factor in the development of gastric retention in the early postgastrectomy period.

In the upright position, ingested barium passes rapidly by gravity directly into the jejunum. Coughing, talking and voluntary changes of intra-abdominal pressure cause small spurts of barium to pass quickly through the stoma as a result of compression on the pouch. Normal gastric tone, jejunal peristalsis, changes in the intra-abdominal pressure, diaphragmatic motion and gravity are probably the primary factors in emptying the gastric remnant.

The reasons for the difference in emptying time after gastroduodenostomy as compared to gastrojejunostomy was not ascertained in this study.

It appears that obstruction of the efferent loop is the most common cause of gastric retention following gastrectomy. It is to be recommended that all patients with gastric retention be adequately examined roentgenographically during the symptomatic period, in order that an accurate determination of the site of obstruction can be made.

HENRY J. ROSEVEAR, M.D.

Use of Y. Plasty for Obstruction of Vesical Neck. Sanatra, T. J., J. Urol. 77:614, 1957.

The treatment of contracture of the vesical neck by open operation via the retropubic approach has been popularized of recent years, especially in the treatment of children.

The author of this paper modifies the Bradford Young procedure of transverse closure of a vertical incision over the anterior prostatic capsule and bladder, by making a Y incision. He had used this operation in 6 cases, the patients being adults with contracture of the vesical neck or median bar, and is satisfied with the results.

SHEPARD JEROME, M.D.

Results of the Treatment of Cancer of the Breast at the Montreal General Hospital. Richards, T. A., Palmer, J. D., and Martin, S. J., Surg., Gynec. & Obst. 104:451, 1957.

The unfavorable present status of carcinoma of the breast has stimulated efforts to obtain better results. The authors, working in the Montreal General Hospital tumor clinic, have recently introduced the method of Richards and Janes of the Toronto group, who have been utilizing high doses of preoperative irradiation followed by radical mastectomy for "locally advanced" operable carcinoma of the breast. The authors have arbitrarily selected this more radical attack on mammary carcinoma in preference to super-radical surgical procedures like those devised by Urban, Wangensteen and Erhenhaft.

Others, notably Delarue and Pisani, independently have published five-year and ten-year survival rates for "operable carcinoma" after using preoperative irradiation therapy in high dosage, and the results have shown a definite improvement over those obtained by radical mastectomy, with or without postoperative irradiation.

High dosage preoperative radiation therapy consists of 6,000 to 7,000 r., M.T.D., for two to three months. This therapy is followed in three to six months, varying with the reaction to irradiation, by standard radical mastectomy. Since 1953, 14 patients have been treated in this manner. There has been only 1 death from carcinoma, and the remaining 13 patients show no evidence of persistence or recurrence of their disease. Moreover, a follow-up of these 14 has revealed no untoward reactions during and immediately after the radiation therapy. Most of the trouble has been in the postoperative phase, owing to delayed wound healing. One patient showed severe radiation necrosis of the skin of the chest wall, which required a large pedicle tube graft for reconstruction. The tangential method and increasing technical skill preclude extensive pulmonary fibrosis.

Analysis of the figures recorded after more or less standard methods of treatment has indicated that treatment at any time is of value, since the total five-year survival rate from the onset of symptoms, irrespective of the type of treatment and including inoperable patients, was 68.3 per cent. This appears favorable when compared to the 22.5 per cent rate of five-year survival from the onset of symptoms with no treatment, as reported by Daland. The 35.8 per cent five-year cure rate of 35.8 per cent, cure being determined by the absence of persistence or recurrence of the disease, stimulated the authors to strive for further improvements.

This preliminary experience with the preoperative use of irradiation in high dosage convinces the authors that this method may prove to contain significant value.

THOMAS WILENSKY, M.D.

Adrenalectomy and Hypophysectomy for Advanced Cancer of the Breast. Atkins, H. J. B., Falconer, M. A., Hayward, J. L., and MacLean, K. S., Lancet 1957, 1:489, 1957.

Since 1896 there have been attempts to treat advanced mammary cancer by modifying the endocrine environment—first by removal of the ovaries and, in more recent years, by adrenalectomy and hypophysectomy.

The authors, working at Guy's Hospital, have had the opportunity over the past three years to perform adrenalectomy or hypophysectomy on patients with advanced carcinoma of the breast for whom the possibilities of direct surgical intervention, irradiation, and hormone therapy have been exhausted. Patients in this category were to be discarded only if they were regarded as quite unsuitable for any operation, being moribund.

The technic of adrenalectomy is described, as well as the preoperative and postoperative management, including a basic plan for the administration of cortisone, desoxycortone, salt and antibiotics throughout both stages of the operation. The technic of hypophysectomy is presented, with the preoperative management; the operation and postoperative care are outlined. On the day after the operation 350 mg. of cortisone is usually given, partly by mouth and partly intramuscularly; this is decreased in stages to an oral dose of 25 mg. twice daily by the eighth day.

Patients who have been subjected to adrenalectomy or hypophysectomy are exposed to certain hazards for the rest of their lives. If these dangers are to be minimized, it is essential that maintenance therapy should be most carefully supervised and that the closest cooperation should exist between the hospital and the practitioner.

The standard dosage of cortisone is 25 mg. twice daily by mouth. Occasionally, patients who are going downhill have been temporarily improved subjectively by an increase to 75 or 100 mg. daily.

Though cortisone, 50 mg. daily, provides adequate replacement of the adrenal glucocorticoids, its effect on the metabolism of salt and water is by no means always satisfactory. The disturbances in the electrolyte metabolism can usually be prevented by giving a large quantity of sodium chloride or by the administration of a mineralocorticoid. The authors' patients were given an implant of desoxycortone acetate, 300 mg., before discharge from the hospital.

Sodium chloride in the form of capsules is not given routinely to their patients save in the period immediately following adrenalectomy. It is used, however, to keep the blood pressure within the normotensive range. Patients who tend to have a raised blood pressure are forbidden table salt. Additional sodium chloride was rarely required by the hypophysectomized patients.

To minimize the possibility of epilepsy, phenobarbitone, 30 mg. twice daily, is administered.

Diabetes insipidus, which usually develops within a week after hypophysectomy but may be delayed for as long as a month or more, is temporary and clears spontaneously between the fourth and eleventh month in all survivors. In some instances it was necessary to give posterior pituitary snuff, 40 mg. from one to three times daily.

Thyroid function was assessed by measuring the serum-cholesterol level. With the onset of definite myxedema, a thyroid extract was administered. The maintenance therapy outlined will maintain the patients who are in good health, but with the onset of acute infection or the stress of an accident or an operation, a substantial increase in the adrenal steroids is called for.

An evaluation of the end results in the 60 cases shows that, of the patients who have already died, those who underwent hypophysectomy survived an average of two months longer than did those who underwent adrenalectomy. These results are not considered statistically significant in the conventionally accepted sense. Nevertheless, in the authors' opinion, there is a definite trend in favor of hypophysectomy, and it may well be that with larger numbers of patients significant differences will appear. There may be alternate methods of destroying the hypophysis, such as by proton bombardment with the synchrocyclotron. Further investigation of the relative merits of hypophysectomy and section of the pituitary-stalk section is also planned.

HENRY J. ROSEVEAR, M.D.

Cushing's Syndrome: Preoperative Differentiation Between Hyperplasia and Tumor. Hinman, F. Jr., Steinbach, H. L., and Forsham, P. H., Urol. 77:329, 1957.

Twenty operative cases of Cushing's disease formed the basis of this study of hormonal excretion and roentgen signs.

A level of hydroxcorticoids above 12 mg. a day is suggestive or diagnostic of Cushing's disease. Adrenal hyperplasia produced a significant increase in corticoid excretion. Cortical carcinoma resulted in the highest levels (38.5 mg. a day).

The ACTH stimulation test is diagnostic of adrenal hyperplastic when the level is over 50 mg. a day. Carcinoma produced no rise in corticoid excretion in 2 cases studied after stimulation by ACTH.

The pituitary ACTH suppression test gives variable results and has no practical value.

The paper deals with the roentgenographic differentiation of tumor from hyperplasia. Tomographic study, combined with extraperitoneal pneumongraphic examination, is of more diagnostic help than are plain films. A rounded mass visible on the pneumongrams, together with elevated corticoid excretion, is diagnostic of adrenal neoplasm. A very small adrenal suggests a neoplasm of the opposite side. Displacement of the left kidney below the right is an unreliable sign.

SHEPARD JEROME

Renal Papillary Necrosis: A Clinopathologic Study of 42 Cases. Simon, H. B., Bennett, W. A., and Emmitt, J. L., J. Urol. 77: 557, 1957.

Renal papillary necrosis occurs when there is compression of pyramidal blood vessels by inflammation, especially the presence of diabetes or obstruction of the urinary tract. Of the authors' series of 42 patients with papillary necrosis who came to autopsy, 81 per cent did not have diabetes.

The signs and symptoms of this condition are discussed. The author states that only a few antemortem or preoperative diagnoses have been reported. The disease is usually fatal. It must be distinguished from acute pyelonephritis, calculus, renal tuberculosis and renal tumor.

The gross and microscopic pictures are described. Microscopically it is essentially an infarct with coagulation necrosis. The treatment is supportive; control of infection and diabetes and relief of obstruction are imperative.

SHEPARD JEROME, M. D.

The human understanding is moved by those things most which strike and enter the mind simultaneously and suddenly, and so fill the imagination.

Human knowledge and human power meet in one; for where the cause is not known, the effect cannot be produced. Nature to be commanded must be obeyed: and that which is in contemplation is as the cause is in operation as the rule.

—Bacon

IMPORTANT ANNOUNCEMENT

Contributors of scientific articles for publication in The Journal of the International College of Surgeons from Europe, the Near East and the Middle East should send their articles to:

European Office of the
International College of Surgeons
6-8 Rue de la Confederation
Geneva, Switzerland

A special committee has been appointed to evaluate the papers submitted for publication. The Journal publishes summaries in English, French, German, Spanish, Italian and Portuguese. Summaries of articles should be included in as many of these languages as possible.

AVIS IMPORTANT

Les auteurs d'articles scientifiques destinés à être publiés dans le Journal du Collège international de chirurgiens sont priés d'adresser leurs articles à l'adresse suivante pour l'Europe, le Proche et le Moyen Orient.

Bureau Européen du
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6-8 rue de la Confédération
Genéve (Suisse)

Un comité a été nommé pour l'examen des articles à paraître. Les auteurs sont priés de joindre à leur travail de brefs résumés en français, anglais, allemand, espagnol, italien et portugais si possible.